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23rd ANNUAL SCIENTIFIC and CLINICAL SESSION

September
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Boston, Massachusetts

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MUSCLE CONTRACTION, FATIGUE AND TRAINING

A Review of the Physiologic Literature

KARL HARPUDER, M.D.

NEW YORK

This review consists of four parts, namely: the physiologic chemistry of muscle contraction, the physiology of muscle fatigue, the systemic syndrome of fatigue and the physiology of training. Parts 3 and 4 will be published in a later issue of the ARCHIVES.

I. Muscle Contraction

Only a few decades ago the problems of the metabolism of the striated muscle during contraction and its relation to the mechanical phases of contraction and relaxation appeared well understood. The Nobel prize in physiology was awarded to two investigators for their apparently final and conclusive work in this field.

The conclusions drawn at that time were that the energy for contraction was supplied by an anaerobic breakdown of glycogen to lactic acid. During the recovery period four-fifths of the accumulated lactic acid would be resynthesized by the muscle to glycogen. The energy for glycogen resynthesis would be gained by oxidation of one-fifth of the lactic acid total to carbon dioxide and water. Lactic acid was made responsible for colloid structural changes of the muscle fibers resulting in their reversible shortening. Biochemical analyses of contracting frog's muscle, accounts of energy production and consumption during the chemical reactions mentioned and actual measurement of heat production during isometric contraction and recovery corroborated the hypothesis.

Since then a tremendous mass of new and partly contradictory observations on the biochemistry of muscle contraction have been accumulated. Today it is impossible to give an account on the basis of one relatively simple hypothesis. The subject is extremely complicated and hard to review systematically. It is, at the same time, extremely important and interesting.

Most physiologists follow the classical school of Meyerhof[1] and Hill[2] and consider contraction an anaerobic process, followed by an aerobic recovery phase. The primary source of energy is the breakdown of adenosine triphosphate to diphosphate and finally monophosphate (adenylic acid). A second source of anaerobic energy for contraction is the splitting of phosphocreatine (phosphagen) to phosphoric acid and creatine. The final supply of contraction energy is derived from the breakdown of muscle glycogen. Hexose is first formed in this process and linked with two phosphate molecules. A fission of hexose biphosphate into two triose phosphate molecules follows. The 3-carbon sugars lose hydrogen in several steps and yield phosphopyruvic acid. Phosphate is now removed and four-fifths of the pyruvic acid reduced to lactic acid and resynthesized to glycogen—the whole cycle so far performed without use of molecular oxygen. The remaining one-fifth of pyruvic acid is finally oxidized to carbon dioxide and water. Glycogen resynthesis and oxidation of pyruvic acid take place during the recovery phase after the contraction.

The phosphate for hexose bonds is at least partly derived from the break-

down of adenosine triphosphate and phosphagen. These substances are resynthesized with phosphate liberated from phosphopyruvate.

The energy for contraction is directly produced from nonsugar phosphate bonds. These are resynthesized at the expense of an anaerobic breakdown of glycogen—over a chain of phosphate-bound intermediaries—to pyruvic and lactic acid. The latter is resynthesized to glycogen. The expense of glycogen re-formation and thereby the final cost of contraction are paid for by combustion of pyruvic acid to carbon dioxide and water.

These observations were made mainly on excised frogs' muscle, on minced suspensions of frogs' muscle and on muscle extracts. A similar cycle of energy-producing and energy-consuming processes is present in yeast and yeast extracts. As a matter of fact, it represents probably one of the major routes of cellular carbohydrate metabolism generally.

Although the transformation of glycogen into pyruvic acid is carried out anaerobically, i. e., in the absence of atmospheric oxygen, it nevertheless involves oxidation. Oxidation is not only the introduction of oxygen into a molecule but the removal of hydrogen from its structure (actually the loss of an electron) in complete absence of oxygen. Biologic oxidation of either type needs for its facilitation specific catalysts (ferments) of protein structure.

The substance from which hydrogen is removed is the donator and a ferment or dehydrogenase facilitates the transfer of the hydrogen to an acceptor substance. The acceptor may remain permanently reduced, e. g., when pyruvic acid accepts hydrogen and is reduced to lactic acid. Frequently the reduced acceptor relays the hydrogen to another acceptor of greater hydrogen avidity and returns itself to its former oxidized state. Several reversible hydrogen carriers frequently form a chain or a complete cycle. They may attach themselves to ferments and are then called coferments; or their relationship to their dehydrogenases may be only temporary and loose. In any case, the ferment is specific and works only with one substrate (donator), while the coferment, or acceptor, may link to several ferments.

Today, three systems of dehydrogenation are known to play a role in the carbohydrate breakdown of striated muscle. No details are available on how these systems are interlocked or how and when they act separately or in combination. There are probably differences between different types of activity, between different types of muscles and between animal species.

One chain of hydrogen relays consists of specific dehydrogenases linked closely to reversible hydrogen carriers which are derivates of members of the vitamin B complex.[3] These are nicotinic acid amide and riboflavin. The ferment-nicotinic acid compound takes hydrogen from triose molecules and catalyzes in several steps their transformation to pyruvic acid. The reduced nicotinic acid transfers its hydrogen to a ferment-riboflavin complex and returns itself to its previous condition, ready for further action. Reduced riboflavin can discharge hydrogen and be restituted in two ways. Anaerobically, riboflavin transmits the hydrogen to pyruvic acid, which is then permanently reduced to lactic acid. Under aerobic conditions another, more avid, carrier, cytochrome, takes the hydrogen away from riboflavin. The cytochrome is finally reoxidized by cytochrome oxidase and atmospheric oxygen, the latter actually functioning as the last irreversible hydrogen acceptor with the formation of water. In the final breakdown of pyruvic acid another vitamin B component plays a role as a coferment, although not in a dehydrogenation. Thiamine phosphate acts as a cocarboxylase in the removal of a carbon dioxide group and production af acetaldehyde.

The described course of reactions is one and probably the simplest route

of glycogen breakdown in the muscle, resulting partly in resynthesis and partly in oxidation to the end products carbon dioxide and water. The dehydrogenation of triose to pyruvic acid may involve an additional shifting of hydrogen in a group of four 4-carbon-2-carboxyl acids, namely, oxalacetic, malic, fumaric and succinic acids.[4] The hydrogen is relayed from the substrate to oxalacetic acid, producing malic acid. Further addition of hydrogen to the molecule results in succinic acid by way of fumaric acid. These hydrogen transfers are carried out with the aid of specific ferments and coferments containing nicotinic acid and riboflavin. Succinic acid is oxidized by the cytochrome-cytochromoxidase system to carbon dioxide and water or to oxalacetic acid, the latter ready to function as a hydrogen acceptor again. In the second case the chain of four acids has coferment action; in the first it transports substrate hydrogen only once during its own breakdown. Similarly, the four acids may act as intermediaries (or as coferments) in the oxidation of pyruvic and lactic acid to carbon dioxide and water.

At least in pigeon's breast muscle, the breakdown of carbohydrate can be still more complicated. The catalytic cycle described in the last paragraph can be combined with the hydrogen relaying system of citric acid.[4] Citric acid is a 6-carbon-3-carboxyl acid, which is easily metabolized in muscle and other tissues. In the combined cycle, triose delivers hydrogen to oxalacetate (last paragraph) and yields pyruvate. Pyruvate and more oxalacetate are then synthesized to citric acid. Hydrogen made available during this last oxidative process is accepted by more oxalacetate. Citrate is dehydrogenated in several steps, losing carbon dioxide and probably forming succinic acid (last paragraph). Oxalacetate functions in these transformations again as a hydrogen acceptor. It is able to accept hydrogen readily from many substrates and to donate it immediately to riboflavin-ferment and the cytochrome-cytochrome oxidase system. The hydrogen shifts within the combined citric acid cycle take place with the aid of dehydrogenases (and their coferments).

This is a simplified review of the main features of the carbohydrate metabolism in striated muscle. Why several modi are employed either separately or in combination is not known. The primary source of energy for contraction is not the carbohydrate breakdown but the splitting of adenosine triphosphate and creatine phosphate. The phosphate of these compounds is used to form bonds with hexose derived from muscle glycogen. These carbohydrate-phosphate linkages are finally broken at the pyruvate stage, and the liberated phosphate is utilized for the resynthesis of adenosin triphosphate. A phosphate shuttle also takes place between the creatine and adenosine compounds. Various ferments and coferments catalyze the phosphate reactions.

Although the reported reactions have been proved beyond doubt, partly on excised frog muscle, partly on muscle extracts and partly on minced muscle suspensions, their physiologic importance for the intact muscle, with normal circulation and innervation, of the mammal, contracting in situ, has been seriously contested.[5] The metabolism of the contracting muscle is then considered an aerobic oxidative process, except the first phase, in which the increased tension of the muscle interferes with its blood supply. This first anaerobic phase lasts fifteen seconds in the rat. The substances which supply the oxidative energy for contraction are still unknown; they may be lactate, glucose or hexose monophosphate. A secondary source of oxidative energy may be the ketone bodies,[6] breakdown products of the higher fatty acids. They have been known to be utilized in striated muscle. Their utilization has recently been shown to increase in contracting muscle.

During the first few seconds of electric stimulation of a rabbit's muscle in situ, large amounts of lactic acid and hexose monophosphate appear. With continued stimulation the formation of these substances slows down and phosphocreatine is hydrolyzed. Still later lactic acid and hexosephosphate disappear and phosphocreatine is resynthesized in the contracting muscle. During contraction under anaerobic conditions, lactic acid is formed from glycogen without intermediary phosphate bonds. The main effect of phosphocreatine hydrolysis is a release of base phosphoric acid, being a weaker acid than phosphocreatine. Lactic acid is thereby neutralized. The formation of lactic acid during anaerobic contraction produces about twelve times as much energy as that required for the resynthesis of phosphocreatine. The latter corresponds with the lactic acid disappearing from the muscle, confirming the buffer theory.

An oxidative reversal of anaerobic reactions is too slow to permit a steady state of contractions (i e., a continuous sequence of contractions without accumulation of metabolites (oxygen debt and fatigue), at the rate actually observed. The rate of lactic acid removal from a frog's muscle is 20 mg. per cent per hour. An aerobic steady state has been maintained at the rate of twenty-three twitches per minute. This would necessitate removal of 16 mg. per cent of lactic acid per minute instead of 20 mg. per cent. per hour. Similar discrepancies are present in the rate of creatine phosphate resynthesis observed and expected in the steady state of contracting frog and mammal muscle. Resynthesis of adenosine triphosphate is still slower. The duration of recovery heat in a contracting frog's muscle would theoretically permit a steady state of only one twitch every forty seconds. Recovery heat appears in the absence of oxygen. The recovery heat cannot be caused by oxidative removal of accumulated metabolites. Tracer studies with radioactive phosphorus prove that phosphocreatine and adenosine triphosphate do not act as phosphor-relays in the formation of lactic acid during contraction.

The hypothesis of an aerobic contracting metabolism of the striated muscle has received support from studies of muscle hemoglobin.[7] Muscle hemoglobin is present in large amounts in muscles with powerful slow repetitive action, e. g., breast muscles of flying birds or leg muscles of running animals. Muscles contracting more than three times per second (flying muscles of insects) are rich in cytochrome and poor in muscle hemoglobin. Muscles of weak repetitive action or of powerful action at great intervals contain only cytochrome and no muscle hemoglobin.

Muscle hemoglobin has a molecular weight about one-fourth that of blood hemoglobin. It combines with oxygen much faster than the latter and at much lower pressures. At an oxygen pressure of 40 mm. of mercury, muscle hemoglobin is 94 per cent and blood hemoglobin 66 per cent saturated. Muscle hemoglobin is half saturated at 3 mm. pressure and blood hemoglobin at 22 mm. pressure. The reaction rates of muscle hemoglobin with oxygen are about 1/1000 second (for half-saturation) to 1/100 second for half-desaturation. Muscle hemoglobin will take over oxygen from capillary and venous blood easily and quickly and deliver it easily to cytochrome oxidase, which functions at oxygen pressures of 0.5 to 5 mm. It forms, therefore, a ready and readily replenished oxygen store in the muscle. There is enough of it, e. g., in a dog's heart, to tide the muscle from one contraction to the next even under conditions of extreme effort.

Changes in the oxygen content of muscle hemoglobin can be examined in the intact soleus muscle of a cat by means of a photocell colorimeter. The oxygen store of muscle hemoglobin is utilized during contraction and replenished immediately after relaxation. During a tetanus an increase in

oxygen consumption is observed in less than one-fifth of a second, and 9 to 40 per cent of the oxygen available in the muscle hemoglobin is consumed in the first second when the rate of oxygen disappearance is at its peak. Changes in the blood supply to the contracting muscle are much slower. Oxygen consumption returns to its resting value in ten seconds after the tetanus is stopped. The rapidly increased utilization of oxygen accounts for the greatest part of the energy requirements of the contracting muscle.

In diving seals, muscle and blood analyses showed that muscle oxygen steadily decreases during the first five to ten minutes of the dive.[8] After this period lactic acid begins to accumulate in the muscle, the rate depending on the degree of struggling. There is a drop in glycogen greater than the increase in lactic acid. Muscle oxygen is exhausted while blood oxygen is still fairly high. Apparently the muscle circulation is more or less shut off during the dive. Oxygenated muscle hemoglobin prevents the formation of lactic acid.

It seems to me possible to unite hypothetically the observations of an aerobic and an anaerobic metabolism of the striated muscle. The first mechanism can take care of the energy requirements of the contracting muscle during a steady state. When the effort exceeds the steady state the anaerobic route is called on to produce the energy necessary for the maintenance of muscular work. The latter situation must occur many times daily in the life of wild animals, during strife and flight, and in the life of civilized man when he climbs two flights of stairs or rushes after a bus. Both routes are physiologic; the second supplementing the first. We are used to consider supplementary sources of energy in animal metabolism, i. e., carbohydrates and fats. Supplementary pathways in the metabolism of one substrate appear at least a physiologic possibility.

This assumption is supported in principle by the effects of various ferment-inhibitors on the respiration of excised frog's muscle.[9] The oxygen consumption of a resting muscle is not or is only slightly diminished by a number of substances which markedly reduce or entirely suppress activity metabolism, i. e., respiration in excess of the resting level. This excess may be caused by electric or chemical stimulation of the muscle (e. g., with caffeine). The various inhibitors of the activity metabolism are known to affect entirely different phases of the carbohydrate breakdown, e. g., the cytochrome oxidase system or the pyruvic acid stage, with its ferments, or the initial transformations of glycogen. These experiments on excised muscle can hardly be considered physiologic. They should arouse interest in supplementary metabolic routes for one substrate which may exist during rest and activity as well as for contractions within and beyond the steady state.

The role of electrolytes for the contraction of the striated muscle is not too well known. Magnesium and calcium are essential components of the ferment-coferment compounds active in the various phases of phosphate transfer. Potassium is released by the contracting muscle into the extracellular fluid and the blood stream. The intracellular potassium is apparently easily replenished by serum potassium during periods of muscular rest. Such potassium shifts may also be related to the metabolism of phosphate compounds.

Only the first steps have been made toward a successful correlation of the metabolic changes to the mechanical phases of muscle contraction.[10] It seems certain that the contractile substance is the protein myosin which in vitro form threads showing the roentgen ray diagram of muscle fibers. The myosin molecules have an elongated structure, their extended polypep-

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tide chains being kept in an oriented position by chains of adenosine triphosphate molecules. The latter have been considered as a "rigid template" or as "chemical wedges interposed at the active linkages" of the protein molecule. The protein myosin is, moreover, inseparably attached to, if it is not identical with, the phosphatase which splits adenosine triphosphate. Therefore the contractile substance myosin would by means of its ferment activity break down adenosine triphosphate, the mold responsible for its shape, and simultaneously release its anaerobic energy—both necessary for contraction. Adenosine triphosphate is the only known muscle metabolite to have an influence on the myosin molecule, and myosin will not act as a phosphatase on any other substrate. The reaction is activited by calcium and magnesium ions.

Recent thermodynamic observations[11] generally favor the reported mechanism of contraction. In an isotonic contraction with performance of work the following phases of heat production are present: contraction or shortening heat, maintainance heat, relaxation heat and delayed anaerobic plus oxidative recovery heat. The shortening heat is supposedly derived from chemical reactions during the "folding" of myosin chains; similarly, maintainance and relaxation heat are referred to chemical events in the linkages of myosin molecules with the phosphate bond "wedges" during the unfolding of the protein chains.

These new studies are of the greatest importance and promise. They do not disprove the existence of an aerobic contracting metabolism (in addition to an anaerobic route) in the muscle with normal circulation and innervation.

II. Muscle Fatigue

Fatigue is the inability of a muscle to reproduce on continuous or repeated stimulation the initial tension (isometrically) or to lift the same load over the same distance (isotonically). The appearance of fatigue depends on the intensity, the duration and the frequency of the stimulus, the load in relation to muscle strength and the oxygen supply to the muscle. In the case of electric stimulation it also depends on whether the stimulus is applied directly to the muscle or indirectly to the motor nerve. Many of the conditions of muscle fatigue are known from common daily experience. The importance of the oxygen supply can easily be demonstrated by arresting the circulation to the forearm with a blood pressure cuff and clenching the fist forcefully several times. Fatigue is complete if the muscle cannot contract any more, and it may be followed by a reversible or irreversible contracture of the muscle.

Until recently fatigue has been considered the result only of accumulation of metabolites during contraction.[12] If contractions are carried out or maintained beyond the steady state and sufficient periods of recovery (and resynthesis) are not interposed, lactic acid reaches extremely high concentrations in the muscle and the stores of phosphocreatine and adenosine triphosphate diminish and finally disappear. The accumulation of lactic acid may be beyond the buffer capacity of the tissue, causing acidosis and thereby producing rigor similar to rigor mortis. These changes usually occur before a marked decrease of the glycogen stores of the muscle is noticeable.

This is undoubtedly the common mechanism in the development of muscle fatigue in an active animal or in man. In recent years, however, a number of observations have been made which prove that an entirely different second mechanism of fatigue exists. Whatever the practical importance of the second type may be, its theoretical interest is extraordinary. It concerns only fatigue appearing during indirect stimulation.

If a muscle is stimulated from its nerve until it is fatigued, a reversal of the direction of the current will cause an immediate recovery. If a cat's muscle with intact circulation is stimulated in situ from its nerve at a rate of thirty or less stimuli per second, fatigue does not appear for hours. At a rate of sixty or more per second, the tension of the muscle decreases considerably after thirty minutes or more of stimulation. However, if stimulation is continued at the same rate, fatigue disappears spontaneously and muscle tension increases, although not quite to its initial level. For reasons that cannot be discussed, the period of fatigue has been called the fourth stage and the recovery period the fifth stage of transmission.[13]

During the fourth stage (fatigue) the muscle develops its initial tension on a direct stimulus. The fourth stage appears independently of the load applied to the muscle. If the animal is injected with enough curare to abolish muscle contraction and the injection is timed so that its effect subsides during the fourth stage of a simultaneously applied high frequency stimulation to a motor nerve, fatigue develops even though the muscle does not contract. These observations, together with those of the last paragraph, make untenable an explanation of the fourth stage on the basis of an accumulation of muscle metabolites. Obviously there must be a deficiency in the transmission of the motor nerve impulse to the muscle fiber. The fourth stage is a transmission fatigue, not a chemical fatigue of the contractile elements.

The conduction of an impulse in a motor nerve and the transfer of the impulse to muscle fibers in the end plate are combined with a release of acetylcholine and the appearance of electric potentials. Acetylcholine is present in the bathing fluid of a nerve after its stimulation and in the venous blood returning from a muscle which contracts under indirect stimulation. Direct muscle stimulation does not produce a liberation of acetylcholine. Acetylcholine is obviously released by the end plates, not by the contracting fibers. A close intra-arterial injection of small amounts of acetylcholine causes a tetanic contraction of the corresponding muscle. The effect of motor nerve stimulation and of intra-arterial injection of acetylcholine is enhanced and made repetitive by physostigmine and is inhibited by curare. Physostigmine paralyzes choline esterase, which destroys acetylcholine. Curare increases the threshold of the end plate to acetylcholine. These pieces of evidence put together speak strongly for an essential role of acetylcholine in the propagation of impulses through nerve fiber and end plate to the muscle fiber.[14]

According to recent experiments the destruction of physiologically important amounts of acetylcholine by esterase could occur with a swiftness comparable to that of the electric phenomena appearing on nerve and end plate during excitation. It is reasonable to assume, though entirely unproved, that the release of acetylcholine is a process of similar duration. Possibly acetylcholine changes the permeability of the surface of nervous structures and thereby initiates the bio-electric phenomena of conduction and transmission of an impulse.[15]

In any case acetylcholine is closely enough connected with the transfer of stimuli in the end plates to be called the transmitter substance for the motor impulse. It plays a similar role at all parasympathetic end organs and at all synapses (sympathetic and within the central nervous system).[14]

During the fourth stage of transmission — the fatigue period — the release of acetylcholine at many end plates is diminished below the effective threshold and each stimulus causes a contraction of only a relatively small number of fibers of the whole muscle. The tension of the contracting muscle is low. During the fifth stage, the acetylcholine output (and the acetylcholine

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Recent thermodynamic observations[11] generally favor the reported mechanism of contraction. In an isotonic contraction with performance of work the following phases of heat production are present: contraction or shortening heat, maintainance heat, relaxation heat and delayed anaerobic plus oxidative recovery heat. The shortening heat is supposedly derived from chemical reactions during the "folding" of myosin chains; similarly, maintainance and relaxation heat are referred to chemical events in the linkages of myosin molecules with the phosphate bond "wedges" during the unfolding of the protein chains.

These new studies are of the greatest importance and promise. They do not disprove the existence of an aerobic contracting metabolism (in addition to an anaerobic route) in the muscle with normal circulation and innervation.

II. Muscle Fatigue

Fatigue is the inability of a muscle to reproduce on continuous or repeated stimulation the initial tension (isometrically) or to lift the same load over the same distance (isotonically). The appearance of fatigue depends on the intensity, the duration and the frequency of the stimulus, the load in relation to muscle strength and the oxygen supply to the muscle. In the case of electric stimulation it also depends on whether the stimulus is applied directly to the muscle or indirectly to the motor nerve. Many of the conditions of muscle fatigue are known from common daily experience. The importance of the oxygen supply can easily be demonstrated by arresting the circulation to the forearm with a blood pressure cuff and clenching the fist forcefully several times. Fatigue is complete if the muscle cannot contract any more, and it may be followed by a reversible or irreversible contracture of the muscle.

Until recently fatigue has been considered the result only of accumulation of metabolites during contraction.[12] If contractions are carried out or maintained beyond the steady state and sufficient periods of recovery (and resynthesis) are not interposed, lactic acid reaches extremely high concentrations in the muscle and the stores of phosphocreatine and adenosine triphosphate diminish and finally disappear. The accumulation of lactic acid may be beyond the buffer capacity of the tissue, causing acidosis and thereby producing rigor similar to rigor mortis. These changes usually occur before a marked decrease of the glycogen stores of the muscle is noticeable.

This is undoubtedly the common mechanism in the development of muscle fatigue in an active animal or in man. In recent years, however, a number of observations have been made which prove that an entirely different second mechanism of fatigue exists. Whatever the practical importance of the second type may be, its theoretical interest is extraordinary. It concerns only fatigue appearing during indirect stimulation.

If a muscle is stimulated from its nerve until it is fatigued, a reversal of the direction of the current will cause an immediate recovery. If a cat's muscle with intact circulation is stimulated in situ from its nerve at a rate of thirty or less stimuli per second, fatigue does not appear for hours. At a rate of sixty or more per second, the tension of the muscle decreases considerably after thirty minutes or more of stimulation. However, if stimulation is continued at the same rate, fatigue disappears spontaneously and muscle tension increases, although not quite to its initial level. For reasons that cannot be discussed, the period of fatigue has been called the fourth stage and the recovery period the fifth stage of transmission.[13]

During the fourth stage (fatigue) the muscle develops its initial tension on a direct stimulus. The fourth stage appears independently of the load applied to the muscle. If the animal is injected with enough curare to abolish muscle contraction and the injection is timed so that its effect subsides during the fourth stage of a simultaneously applied high frequency stimulation to a motor nerve, fatigue develops even though the muscle does not contract. These observations, together with those of the last paragraph, make untenable an explanation of the fourth stage on the basis of an accumulation of muscle metabolites. Obviously there must be a deficiency in the transmission of the motor nerve impulse to the muscle fiber. The fourth stage is a transmission fatigue, not a chemical fatigue of the contractile elements.

The conduction of an impulse in a motor nerve and the transfer of the impulse to muscle fibers in the end plate are combined with a release of acetylcholine and the appearance of electric potentials. Acetylcholine is present in the bathing fluid of a nerve after its stimulation and in the venous blood returning from a muscle which contracts under indirect stimulation. Direct muscle stimulation does not produce a liberation of acetylcholine. Acetylcholine is obviously released by the end plates, not by the contracting fibers. A close intra-arterial injection of small amounts of acetylcholine causes a tetanic contraction of the corresponding muscle. The effect of motor nerve stimulation and of intra-arterial injection of acetylcholine is enhanced and made repetitive by physostigmine and is inhibited by curare. Physostigmine paralyzes choline esterase, which destroys acetylcholine. Curare increases the threshold of the end plate to acetylcholine. These pieces of evidence put together speak strongly for an essential role of acetylcholine in the propagation of impulses through nerve fiber and end plate to the muscle fiber.[14]

According to recent experiments the destruction of physiologically important amounts of acetylcholine by esterase could occur with a swiftness comparable to that of the electric phenomena appearing on nerve and end plate during excitation. It is reasonable to assume, though entirely unproved, that the release of acetylcholine is a process of similar duration. Possibly acetylcholine changes the permeability of the surface of nervous structures and thereby initiates the bio-electric phenomena of conduction and transmission of an impulse.[15]

In any case acetylcholine is closely enough connected with the transfer of stimuli in the end plates to be called the transmitter substance for the motor impulse. It plays a similar role at all parasympathetic end organs and at all synapses (sympathetic and within the central nervous system).[14]

During the fourth stage of transmission — the fatigue period — the release of acetylcholine at many end plates is diminished below the effective threshold and each stimulus causes a contraction of only a relatively small number of fibers of the whole muscle. The tension of the contracting muscle is low. During the fifth stage, the acetylcholine output (and the acetylcholine

content of the conducting nerve) increases above the threshold value in most end plates. The contraction strength improves because more fibers contract. With a slower rate of stimulation (less than thirty per second) a steady synthesis and release of acetylcholine are apparently maintained for hours.

We have therefore to separate a chemical fatigue of the contracting muscle fibers and a transmission fatigue of the end plates in the contracting muscle. The number of electric discharges recorded during an active contraction in a single motor nerve fiber averages ten to twenty per second and is probably too low for transmission fatigue. Rates up to a hundred per second have been observed in extensor nerves.[15] The question of transmission fatigue during spontaneous activity is entirely speculative. This does not diminish the significance of the phenomenon. The physiology and pathology of muscle contraction have to be supplemented by the physiology and pathology of muscle excitation.

The fatigability of a patient with hyperthyroidism seems to be on the basis of depletion of energy stores in the muscle and a wasteful resting-and-contracting metabolism. The fatigability of a patient with myasthenia gravis, however, is caused by a deficiency in transmission — an insufficient release of acetylcholine or an increased threshold of the muscle synapse or a combination of the two.[16] In a denervated muscle, changes in the storage of energy materials as well as changes in the mechanism of excitation occur. Electric stimulation of the severed distal nerve stump depletes its acetylcholine content, and its resynthesis is slow.[17] Electric stimulation increases the energy stores of the muscle. Therapeutic muscle stimulation in man — which always involves the motor nerve — is still a measure open to discussion. A degenerating motor nerve contains less than the normal amount of acetylcholine, while the esterase content of the end plates remains unchanged. If the acetylcholine content of the end plates runs parallel to that of the nerve fibers — a reasonable assumption — there will be a discrepancy between the transmitter substance and its destroying agent. This would explain the increased fatigability of the denervated muscle and the lack of response to rapid indirect stimulation.

Electrolytes play a certain role in transmission and transmission fatigue. Potassium ions are released simultaneously with acetylcholine. Potassium may facilitate transmission or depress it, according to its concentration and the responsiveness of the synapse.[18] It probably is of importance in the turnover of the inactive acetylcholine precursor and free acetylcholine.[19] It has been therapeutically used in myasthenia gravis. In familial paralysis, potassium is acutely diminished in the blood serum with the onset of a paralytic attack, which can be abolished by the administration of potassium. Paralysis can be produced in dogs by the injection of desoxycorticosterone acetate, which results in a marked reduction of serum and muscle potassium. The administration of potassium salts is curative. The presence of calcium ions is essential for the synthesis of acetylcholine.[20]

Briefly it may be mentioned that the pharmacologic or therapeutic effect on striated muscle of substances such as curare, quinine, guanidine and ephedrine is based mainly on their action on the end plate and transmission.

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FROSTBITE *

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As a result of the need for troops in all branches of the service to operate in the arctic regions and in subzero weather, both the prevention and the treatment of frostbite demand much in the way of experimental investigation and recommendation, not only on the part of the medical departments of the armed forces, but on the part of physicians on the home front. The operation and maintenance of mechanized equipment in extreme cold create added hazards.

A brief historical review will show that failure to prevent the incapacitation of troops from frostbite may result in morbidity high enough to impair the progress, if indeed not the loss, of an entire campaign.[1] Napoleon's campaign in Russia was a total failure owing in no small measure to inexperience with the prevention and treatment of frostbite. In World War I the blizzard of November, 1915, is reported to have caused approximately 16,000 instances of frostbite in Gallipoli and about 1,000 instances in Serbia. Greene[2] predicted correctly that if the Germans were forced to dig in for the winter of 1941-1942, frostbite would again be a potent natural ally of the Russians. Much evidence has been accumulated to prove that there was great morbidity among the Germans from frostbite. Although the Germans were more experienced and better prepared in the winter of 1942-1943, the problem of frostbite was still of great importance.

So that the United States troops might be instructed in the prevention of frostbite, a basic field manual[3] dealing with operations in snow and extreme cold was published by the War Department as early as September, 1941. This manual was of great help to those forces which were sent to arctic defense posts in the haste which was so urgent.

It has been shown by Bazett[4] and others that immersion of the forearm in cold water diminishes the rate of circulation so markedly that eventually even comparatively deep tissues may have a temperature little above that of the water itself. According to Lewis,[5] with the first exposure of the skin to cold, three separate stages of reaction occur. First, there is a direct local vasoconstriction which takes place in the superficial vessels and which persists. Second, there occurs an immediate general vasoconstriction by reflex action through the central nervous system. This is transitory and may be demonstrated by cooling the limb with a tourniquet applied. Third, the cold venous blood returning from the cooled tissues joins and lowers the temperature of the blood in the general circulation. The blood travels to and acts on a central nervous mechanism which is very sensitive to slight changes in cold. The response is in the nature of a general vasoconstriction which is persistent. It has been pointed out by Bazett that, although cold usually causes vasoconstriction, extreme cold may produce dilatation of some arterioles and capillaries in the skin of the extremities. Such a reaction is produced only when the temperature of the skin is below 64.4 F. The reflex that causes this is probably akin to a mild inflammatory reaction. This reaction may act to protect the peripheral areas from further injury. Krusen's textbook[6] on physical medicine contains much information on the physiologic effects of cold. It includes a complete investigation into the observations of other workers. Krusen said that phagocytosis

* This work was done by Major Bankert as a student officer, under the direction of Dr. F. H. Krusen, while he was on assignment at the Mayo Foundation for instruction in physical medicine.

and processes of immunity are unquestionably delayed in the regions which are exposed to local cooling. Local metabolism and the local volume of blood are diminished by application of intense cold. A marked decrease in the systemic temperature tends to increase the number of leukocytes and the amount of sugar in the blood. He further stated that shivering which results from cold may cause an increase in metabolic activity and a rise in the previously lowered body temperature. A temperature of 50 F. or lower gradually benumbs the skin. Fine touch and ultimately all touch and pain sense are lost. The muscles of the extremities, especially those of the hand, become weaker owing to cooling of the muscles and motor nerves. This interferes with local movement and hence with fine control of movement. Function of the member is seriously impaired. When the temperature is further lowered, the freezing point of skin is reached at various levels. The true freezing point of skin according to Lewis[7] is between 28.5 and 32 F. However, he further observed that freezing of the skin rarely occurs at the true freezing point because of the phenomenon of supercooling, which is the capacity of the substance to withstand temperature below its ordinary freezing point without solidifying. This phenomenon differs in degree in different skins, in different areas of the same skin, in the same skin, in the same areas on successive observations, in length of exposure and in other factors. Freezing usually occurs between 25 and 14 F. Soaking the skin in water will abolish the capacity for supercooling. Skin which has been bathed too often will not supercool well. Certain oils, notably whale oil, cod liver oil, olive oil and castor oil, when applied to the skin give it the capacity to supercool. After supercooling has occurred, freezing with crytallization spreads throughout the involved tissues. Frostbite occurs most commonly at temperatures below 14 F., especially when a moderate to strong wind is blowing. Exposure of the skin to cold metal will produce freezing at higher temperatures.

Thawing of frozen tissues is the period of greatest danger. During thawing, a red area surrounds the involved region, gradually invading it until the white skin becomes red. A wheal usually develops owing to the local liberation of a histamine-like substance. This phase is accompanied by itching. In moderately severe frostbite, the redness and swelling of the involved region and tenderness and pain on warming usually occur on the day after the frostbite was incurred. If the involvement is mild, peeling of the skin frequently occurs and the skin may be pigmented for months. If the frostbite is moderately severe, a blister may form and burning pain may occur. If the frostbite is severe, necrosis occurs and there is extravasation of whole blood into the tissues. Consequently, the skin becomes darkened or ecchymotic. The sudden production of edema under pressure during the thawing period is responsible for a great degree of the serious damage, owing to splitting and separation of the intercellular and intracellular tissue substances. This edema also interferes in a mechanical way with the blood supply in such a manner as to cause local tissue anoxia and gangrene. Pathologically, edema of the epidermis and dermis occurs, and there is marked perivascular infiltration of the dermis with lymphocytes, erythrocytes and polymorphonuclear leukocytes. Thrombosis of the vessels may be seen. Even in those cases in which there is only superficial involvement of a limb, the appearance is often such as to cause one to give guarded prognosis. At times, pulsation in the involved extremity is so weak that one is almost convinced that the blood vessels are completely blocked. The necrotic tissues may turn black and later peel off, leaving an appearance of the skin similar to that commonly seen after the tannic acid treatment of burns. Disturbances such as various degrees of hyperesthesia, hypoesthesia and paresthesia are common after severe frostbite of the extremities.

Education of military personnel in the recognition of the symptoms and signs of impending frostbite is of primary importance. In the case of frostbite, it is certainly true that "an ounce of prevention is worth a pound of cure." One of the most important signs of impending frostbite is the appearance of a dull yellowish pallor. This is associated with either numbness or a prickling sensation. Very quickly, often in a matter of a few minutes, the skin will appear to have a dull waxy white appearance. There may be either absolute loss of sensation or increase in the prickling sensation. Often when a group of men are actively engaged they will not become aware of impending frostbite themselves, but an officer or soldier familiar with the symptoms and signs will take notice and also take the necessary steps to prevent a severe reaction.

A number of factors may hasten the production of frostbite or aggravate the resulting damage. A knowledge of these factors is important in the prevention of this condition.

Dampness must be avoided. Any activities which tend to cause excessive perspiration must be avoided. In extremely cold climates people must learn to live a slow life, so to speak. Work must proceed slowly.

Circulatory stagnation must be carefully guarded against. Tight clothing of any sort interferes not only with blood flow but with the proper evaporation of perspiration.

Wind at high velocities will penetrate clothing and induce freezing at relatively high temperatures by causing rapid evaporation of perspiration and by blowing the layer of warm air away from the body surface.

Anoxia, whatever the cause may be, certainly increases the susceptibility of the tissues to damage from cold. When the available oxygen is deficient, the blood supply to the skin becomes reduced because the blood is shunted to the deeper organs. A vicious circle therefore is established. The warming effect of oxygen at high altitudes is well known, and its value in frostbite also is known. In addition to reduced atmospheric oxygen pressure, which occurs in modern aviation, other factors may produce anoxia and must be combated. These included anemia irrespective of its cause, stagnation of the circulation and poisoning by chemicals such as might occur accidentally or from their employment in chemical warfare. After surgical anesthesia great care must be taken to protect the patient from cold until all of his metabolic processes are restored to as nearly normal as is possible. Greene[2] recommended the use of whole blood rather than plasma, when transfusion is necessary, to increase the oxygen-carrying capacity.

Malnutrition may increase the danger of frostbite in several ways. It is best to eat at least four times daily a diet which is well balanced. Fruit juices seem to be required. Warm tea is the drink of choice and is the one most commonly used by persons who live in the subzero regions of Canada. Alcohol in any form is dangerous and should be avoided. I have never seen any need for it therapeutically, although this statement may be questioned. Any factors which may operate to produce edema must be eliminated, since edema predisposes to the development of frostbite.

Warmth during the period of thaw following frostbite is potentially dangerous. Ever since the observations of Larrey, Napoleon's surgeon general, heat in the treatment has been recognized as dangerous. Most certainly among the Eskimos and experienced explorers, who are known not to employ warmth, frostbite rarely occurs. It has been shown that even moderate warmth may be harmful. Smith, Ritchie and Dawson[8] demonstrated that after tissues are damaged by cold, exposure to water at a temperature of 98.6 F. will produce edema or greatly aggravate an existing edema. Lewis

and Love[9] showed that this edema is injurious and capable of temporarily obstructing some of the arterial blood supply to the tissues. Greene[3] reported that gentle warmth greatly increases the total tissue lost after freezing.

Trauma is certainly a factor in producing frostbite. Rubbing an involved part with snow or ice must be avoided. Massage with a coarse towel or pinching the skin only tends to increase the damage and the risk of infection according to Andrews[10] and Greene.[11] Even small lesions must be meticulously handled. It has been found that the morbidity will be markedly reduced if men with small sores and abrasions are relieved from duty involving exposure until the leisons are completely healed. A frostbitten hand should be carried in a sling or against the body in such a manner as to protect it from the wind or cold. When the feet are involved, the footgear should be loosened carefully and cut off if necessary and the patient carried by litter or other suitable means to shelter or to the place where he is to be treated.

The proper type of clothing and equipment and the knowledge of their proper use will do much to eliminate the occurrence of frostbite except under the most severe conditions. The office of the surgeon general, through its various agencies, has been active in developing and improving clothing and equipment for troops serving in subzero temperatures. Indeed, much work is being done at this very time to improve what was first developed for general use. No matter how good the footwear, long marches in cold weather are always potentially dangerous because of chafing and sweating. Clean dry socks are absolutely necessary to prevent cold feet. At least two pairs of socks should be worn, and these should be of coarse weave so as to allow evaporation of perspiration. Shoes must not fit too snugly and must be kept dry. A change of shoes is a necessity to permit the shoes to dry out. Rubber boots or oil-treated boots or shoes should not be used except where they are needed to keep the feet from becoming soaked with water. Felt boots or shoes are fine, but oversocks or slippers made of duffle similar to the wool of the famous Hudson Bay blankets are far superior. The warmest footgear known is certainly the Eskimo "mukluk." Usually the outer layer is waterproof sealskin, with the fur sides turned out. Caribou skin socks are worn inside this with the fur side turned inward, and inside this is either a fine grade wool sock or another caribou skin sock of young caribou hide.

Gloves are an important item. The best type is a gauntlet mitten which has a detachable inner lining such as duffle. The lining should be washed and dried frequently. The gauntlets of the mittens should fit so that snow or sleet cannot get in but perspiration can escape. The mitten itself may be made of deer skin, moose skin, caribou skin or even sealskin. The cuff or edge of the mitten should have a drawstring, and it is well to line the edges with wolverine fur.

Clothes should be loose and light in weight, and underwear should be 100 per cent wool. Underwear, like socks, must be kept clean and changed frequently. Clothing should be worn in layers to take advantage of the principles of insulation and to allow the proper escape of perspiration. The outside of the outer garment should be covered with a layer of Grenfell or Byrd cloth. The hood of the parka (outer garment) should be long enough to protect the face and should be fitted with a drawstring to narrow the opening. The edge of the hood is best lined with wolverine fur. Moisture from the breath does not collect on this fur and freeze. Zipper fasteners and other metal fasteners should be avoided in these garments, especially where there is any danger of their coming into contact with the skin.

There is no unanimity of opinion concerning the wearing of beards. There is no question but that the moisture of the breath collects on the beard and freezes. However, men who live in extremely cold temperatures seem to prefer to wear a beard (and a long one at that) instead of shaving. They get along well in spite of the fact that the beard does interfere with eating and smoking.

Treatment

The treatment of mild frostbite is simple. When a white patch occurs on the face or ears one should cover it with a soft mitten, cease motion and, if possible, get out of the strong blasts of wind. It is advisable to warm the affected part with the uncovered hand. If the hand is involved, it is best to get the hand as close to the skin of the body as possible by inserting it through the garments or into the trousers in the region of the groin. If a foot is involved, the best thing to do is to remove the footgear and socks and place the affected member next to the skin of another person. A very gentle type of centripetal massage may be used, but vigorous rubbing is absolutely wrong. Warmth greater than body temperature should *never* be applied. If a part is not quickly restored to normal, it should be wrapped in dressings which are sterile, or as clean as possible, covered loosely but warmly and placed at complete rest. The application of antiseptics to the involved skin is to my mind a questionable procedure and not worth the effort. If the affected person has not received adequate doses of tetanus toxoid, or if further antitetanic treatment is deemed advisable, such treatment should be given, since it has been shown that tetanus is a common and serious complication of frostbite. The patient should be given warm drinks, especially tea, and warm food, and he should be covered with additional clothing to prevent further exposure, after which it is desirable to place him in a shelter in which the temperature is about 60 to 65 F. The affected part should be elevated about 6 or 8 inches and allowed to warm gradually to room temperature. Unless infection sets in, there is no need to disturb the dressings. In the treatment of moderately severe or severe frostbite, the aforementioned basic principles should be followed. Certainly if there is no evidence of infection, the dressings should be left alone for ten to fourteen days. This will prevent the added risk of infection. If edema occurs, moderate refrigeration, such as that employed in the treatment of immersion foot, is advisable. In the presence of spreading infection, incision and drainage, with sulfanilamide used locally and one of the sulfonamide compounds used internally, certainly are indicated. According to the work and conclusions of Lewis,[5-7] it may also be desirable to employ cooling of a part when pain accompanies the approach of the environment to room temperature. Shipley and Yeager[12] have expressed the belief that suction pressure has great possibilities in the treatment of frostbite. Kaplan[13] has reported that good results may be obtained by the local application of ultraviolet radiation of sufficient intensity to produce erythema of second degree. This treatment is repeated when the reaction subsides. Krusen recommended the use of the whirlpool bath to rehabilitate the involved limb. This would no doubt cause an increase in the circulation through the involved tissues. It would tend to relieve disturbances of pain sensation, which are frequent sequellae, and would prepare the part for subsequent massage and passive or active assistive movements.

Conclusions

I am sure that there has been a great deal of work done on this subject by investigators both here and abroad, especially by the Russians and Ger-

mans, which has not as yet been published or released for general knowledge. Of course I wish to apologize for not having covered all the literature which may possibly be available dealing directly or indirectly with the subject. As a result of my experiences I have noted that hoarseness or even aphonia may develop rapidly as a result of sudden inhalation of cold air through the mouth. One must learn to breathe through the nostrils. There are persons whose feet and hands perspire readily and in whom one may find evidences of vasomotor instability or disturbance of the autonomic nervous system. These persons do not stand cold climates and cannot adapt themselves to military service in subzero temperatures, particularly if they work under great tension and are unable to relax readily. Finally, I wish to state that the investigations which are now being carried out will further our knowledge of the effects of cold and thereby enable us to improve preventive treatment. There is no doubt that more will be accomplished to improve our knowledge of cryotherapy.

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REDUCTION OF MANPOWER LOSS FROM GONORRHEAL URETHRITIS BY EARLY APPLICATION OF FEVER-CHEMOTHERAPY *

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The conservation of manpower has become the cry of a nation at war, and any unnecessary loss of time is definitely hampering to the war effort. Military tactical experts have learned that for each casualty concerned they must plan on an average of two or three normal men detailed for his proper care. In this war, in which civilian men and women are vital to the production lines, a similar trend of thought relative to total manpower jeopardy is easily conceived.

Gonorrheal infection has proved itself beyond doubt to be one of the distinct hazards to manpower efficiency in the armed forces. Military literature is replete with the concern of those charged with the responsibility of this problem. An article published in May, 1943, by the American Neisserian Medical Society stating that several million new cases occur each year bears witness to the assumption that civilian defense is sharing the manpower loss from this scourge.

Analysis shows that the incidence is greatest in the age groups most vital both to combat duty and to the civilian production lines. The persons affected cannot be economically spared from duty for long periods without seriously hampering the war effort. Therefore, any management which promises to shorten the convalescence period warrants full consideration.

Methods of Study

In the military services it is customary on the discovery of an infection to hospitalize the patient until he is bacteriologically and clinically well. This affords opportunity for study under uniform conditions of the total loss from duty and the comparative effectiveness of various forms of therapy.

In the twenty months ending Aug. 1, 1943, 1,783 patients with a diagnosis of gonorrheal urethritis were admitted to the venereal wards of one of the five largest of the United States naval hospitals. Four types of therapy were used, but all patients received first a trial treatment of the conventional type, and only those in whom this failed received one of the three adjuncts. Since the disease in all the patients who received fever therapy was resistant to primary therapy, 25 patients from our civilian practice who received artificial fever-chemotherapy as the primary treatment are reported on for comparison.

Conventional therapy consisted of the oral administration of sulfathiazole, 1 Gm. (15 grains) six times daily, over a period of one to two weeks, according to whether or not the drug had been given prior to admission. Fluids were forced to the point of assuring a dilute urine at all times. While

* The opinions and assertions expressed herein are the private ones of the writers and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

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the majority of patients were ambulatory, all strenuous activity was stopped. No mechanical instrumentation was utilized during the positive infectious stage and no prostatic massage until after the second glass of urine had become clear. Local therapy by anterior urethral instillations was given in selected cases but only when the infection was confined to the anterior portion of the urethra. A majority of all patients received either daily hot sitz baths or short wave diathermy by induction seat coil.

From the group failing to respond to this conventional type of treatment, in many cases after repeated trials, 269 patients were segregated into subgroups and given one of three types of adjunct therapy. One group (50 patients) received foreign protein consisting initially of 0.3 cc. of triple X (Navy) typhoid vaccine intravenously; repeated doses up to 0.6 cc. were administered to obtain the temperature and number of sessions (three to five) desired.

A second group (116 patients) were given fever therapy by physical means at low temperature (104 F.) for four hours, each patient receiving six sessions. Sulfathiazole was continued during this period. The third group (103 patients) were subjected to fever therapy at high temperature (106 F.) for four hours, each patient receiving two sessions, in combination with 7 Gm. (105 grains) of sulfathiazole, which was started at 12 noon on the day prior to fever and given in divided doses until midnight of that day. This sustained a blood level of between 9 and 12 mg. per hundred cubic centimeters.

Daily study of progress, including smears and two glass urine tests, was routine in all cases. Observation for seven to ten days after a negative bacteriologic examination of the urethra, prostate and urine was instituted before the patient was discharged to duty.

Brief tables to avoid excessive detail have been constructed to emphasize the manpower loss involved and a possible remedy for that loss.

Technical Aspects of Treatment

The conventional routine, together with the technic of foreign protein therapy, are well established and need no further elaboration. It is the fever therapy in combination with sulfonamide compounds with which we are mostly concerned. In this connection, the premise that the department must be supervised and manned by specially trained personnel is basic.

Prefever management consisted of a physical study of the patient, electrocardiogram, roentgenogram of the chest, complete blood count and urinalysis. This is desirable in industrial or governmental practice, where these adjuncts can be routine and at a minimum or no cost to the patient. In civilian practice, however, where unnecessary expense must be curtailed, these tests are not all required if a competent physician is in charge of the physical study. The vast majority of the patients are excellent "fever risks."

On the day prior to fever treatment fluids are given freely; protein, chlorides, dextrose and milk are emphasized in the diet, and at 8 p. m. the patient receives a soapsuds enema.

Breakfast is withheld on the morning of treatment, and just prior to induction of fever 8 ounces of 50 per cent dextrose lemonade and 2 Gm. (30 grains) of calcium gluconate are given orally. This seems to reduce both the exhaustion and the tendency toward tetanic manifestations often encountered. Chlorides and water are given freely during the session. The rectal temperature is elevated gradually to the desired level and maintained for four hours, after which it is allowed to recede to 101 F., when a sponge bath is given and the patient returned to bed until the following morning. The session is repeated within four days. Sedation is usually required during the high temperatures, and after experience with many, we prefer spasmalgin (Roche) ampoules, given hypodermically.

Any standard accepted apparatus for production of fever is suitable if the department is provided with proper supervisory and technical skill.

Results and Analysis

Tables constructed to reveal all details of the study would be too long to be practical, and therefore brief ones sufficient to emphasize the scope are included. Table 1 reveals a mean average of 62.5 days from admission of

TABLE 1. — *Comparative Effect of Various Types of Therapy on the Reduction of Manpower Loss Caused by Gonorrheal Urethritis.*

Total Patients	Type of Therapy	Average Hospital Days Per Patient for Group	Range of Hospital Days Per Patient
1,783	Conventional routine	62.5	15 to 203
50	Routine (failure), then foreign protein, triple X (Navy) typhoid vaccine	47.3	18 to 167
116	Routine (failure), then chemotherapy and fever, 104 F., 4 hours, 6 sessions	40.2	15 to 91
103	Routine (failure), then chemotherapy and fever, 106 F., 4 hours, 2 sessions	22.1	12 to 53
25	Immediate chemotherapy and fever, 106 F., 4 hours, 1 to 2 sessions	11.0	8 to 14; 1 patient failed to respond in 2 sessions

the patient until his return to duty for the 1,783 patients receiving routine conventional therapy. This figure is conservative, since the sick days of those in whom this therapy failed and who received adjunct therapy were not charged against the group receiving conventional treatment after such therapy was instituted. This figure coincides closely with those of two other reports from different localities; of these one gave a mean average of 72 days, and the other, 52 1/3 days. Some curtailment of sick days by the foreign protein and fever therapy at low temperatures is evident; there was a further drop with chemotherapy, and fever at higher levels, and there was a dramatic reduction with the combined therapy used in early cases.

Tables 2, 3 and 4 represent a more detailed breakdown of individual

TABLE 2. — *Analysis of 53 Cases of Resistant Gonorrheal Urethritis Over a Period of Three Months, Showing the Average Number of Sick Days Per Patient.**

Month, 1943	Total Patients	Average Total Sick Days Per Patient	Average Days Before Fever Per Patient	Chemotherapy-Fever Treatments, 106 F., 4 Hrs.	Average Days After Fever Per Patient
March	9	72 1/5	51	2	10 1/3
April	21	53 1/7	35 1/7	2	14 2/3
May	23	43 1/7	28 5/7	2	8 2/3
Three Months Total	53	56.22	38.28	..	11.22

* The unnecessary increase in hospitalization time is emphasized by a comparison of the number of days before and the number after chemotherapy and fever therapy at high temperatures.

TABLE 3. — *Analysis of 9 Cases of Resistant Gonorrheal Urethritis Treated With Chemotherapy and Fever Therapy at High Temperatures.**

Cases	Total Hospital Sick Days	Sick Days Before Fever Started	Number of Chemotherapy-Fever Treatments	Sick Days After Fever	Comment
1	51	33	2 at 106 F., 4 hrs.	12	The days consumed in
2	214	194	2 at 106 F., 4 hrs.	16	observation and
3	112	92	2 at 106 F., 4 hrs.	4	clerical procedure
4	35	24	2 at 106 F., 4 hrs.	6	necessary to return
5	41	25	2 at 106 F., 4 hrs.	12	men to duty
6	43	33	2 at 106 F., 4 hrs.	4	were eliminated from
7	38	15	2 at 106 F., 4 hrs.	12	the statistics, since
8	92	80	2 at 106 F., 4 hrs.	7	they were not uniform
9	69	63	2 at 106 F., 4 hrs.	13	in any group

* Note the greater reduction in number of sick days in these cases as compared with those in table 4, in which low temperatures were induced.

TABLE 4. — *Analysis of 9 Cases of Resistant Gonorrheal Urethritis Treated With Chemotherapy and Fever Therapy at Low Temperatures.**

Cases	Total Hospital Sick Days	Sick Days Before Fever Started	Number of Fever Treatments	Sick Days After Fever	Comment
1	96	30	5 at 103-104 F.	54	Each treatment was
2	57	18	6 at 102-103 F.	24	maintained for at least
3	134	86	5 at 103-104 F.	38	4 hours; the days in-
4	94	28	5 at 103-104 F.	54	volved in clerical and
5	105	59	10 at 102-103 F.	13	observation technicali-
6	119	44	5 at 102-103 F.	64	ties, prior to patient's
7	88	46	4 at 103-105 F.	35	return to duty were
8	186	95	10 at 102-104 F.	23	eliminated owing
9	103	83	5 at 103-104 F.	10	to lack of uniformity

* Note by reference to tables 1 and 3 the comparative lack of effect in the reduction of sick days with this technic.

cases and emphasize, first, the manpower loss with the use of routine therapy prior to the institution of adjunct treatment; second, the comparative shortening of convalescence by the adjunct therapy, and, third, the effectiveness of the lower and higher temperatures in bringing about this reduction.

Comment

It should be emphasized that the manpower loss indicated by this study must be multlpiied by thousands for a proper estimate of the relation it bears to the incidence of total gonorrheal infections existing in the civilian and armed forces of this country. Obviously, when considered in this light, the problem reaches proportions of great magnitude. Reductions of twenty days or 20 per cent may become staggering figures when considered in terms of these multiples.

No arbitrary time limit has, as yet, been set for classifying the treatment of gonorrhea—by any particular method—as a failure. In military service, where manpower loss is always a concern and where methods are available for shortening convalescence, it would seem that fifteen days should be the average period of treatment before the patient is returned to duty.

Vital to the subject is the fact that fever therapy apparatus at present is well distributed throughout the Navy, Army and United States Public Health Services. Furthermore, these services have been conducting extensive training in both medical and technical skill to assure proper management.

The cry of danger, while possibly justified in the past, should in the light of present knowledge be modified. Fever therapy has been tested over a period of fourteen years and should now assume its proper position in scientific medicine. Patients such as those under discussion represent excellent risks when compared with those encountered in general practice. Only two moderately severe and one severe reaction were encountered during this study and there were no fatalities. This result compares well with that of any other established procedure in medicine or surgery involving a similar degree of skill. These statements should not be misconstrued as belittling the highly specialized requirements necessary for successful fever departments, but they serve to refute unfounded opinions, too generously expressed by the inexperienced, that fever therapy invariably spells last resort and often death.

Controlled study has convinced us that artificial fever and chemotherapy combined are superior to either alone, although the treatment itself is more difficult technically. When two sessions at 106 F. for four hours each will produce the results shown, both conservatism and safety suggest these in preference to a single treatment at 106.8 F. to 107 F. for ten hours. In this connection we are not in agreement with those who claim that the ther-

mal death point of the gonococcus is the only curative factor involved; numerous patients have been cured at temperatures below the lethal point.

The contention that there is a sufficiently high percentage of cure with chemotherapy alone does not coincide with our experience or with that of shipmates at sea. Ever increasing failures, together with many reactions to the sulfonamide compounds, are coming to light. As a matter of fact, the vast majority of the cases composing this report are just such failures, and they represent no small number for a single admitting unit.

Summary and Conclusions

An analysis of 1,808 cases of gonorrheal urethritis, in 25 of which artificial fever-chemotherapy was given during the early acute stage, is presented with respect to manpower loss during active warfare.

The relative merits of four types of therapy are shown.

Combined chemotherapy with fever therapy administered in two four-hour sessions at 106 F. is shown to be superior to the other methods studied.

Artificial fever-chemotherapy when possible should be given early in the infection.

The safety of the combined management is discussed.

SOME COMPLICATIONS OF THERAPEUTIC HYPERPYREXIA* †

LIEUTENANT COMMANDER HARRY S. ETTER
(MC) U. S. N.

Artificially induced fever has attained a definite and important place among modern therapeutic procedures. Its value in the treatment of sulfonamide-resistant gonorrhea, syphilis of the central nervous system, brucellosis, Sydenham's chorea and possibly rheumatic fever is well recognized. It has been employed for the treatment of more than fifty different pathologic conditions, but, as pointed out by Krusen,[1] it has been of little value in the majority of these conditions.

The use of hyperpyrexia is attended by many actual as well as potential dangers, as is usually the case with any powerful therapeutic agent. Any one administering fever therapy should be able to recognize the resultant complications readily and should have a thorough understanding of their pathology, prevention and management. Several of these complications will be discussed in the light of recent investigative work relative to their pathologic physiology and the rationale of some of the measures used to prevent them. Specific methods of treatment will be touched on when necessary. Fatalities have occurred in the past and undoubtedly more will occur in the future, but with the improved methods now being used for the induction and maintenance of the fever, and with a better understanding of the physiologic changes taking place in the body while it is undergoing high temperatures, these fatalities should and can be reduced to a bare minimum.

In an attempt to obtain a better insight into the problem, the pertinent

* This work was done by Lieutenant Commander Etter while he was a student officer, under direction of Dr. F. H. Krusen, on assignment at the Mayo Foundation for instruction in physical medicine.

† The opinions and assertions contained herein are the private ones of the writer and are not to be considered as official or as reflecting the views of the Navy Department or the naval service at large.

findings at necropsy of several patients who died after fever therapy will be reviewed briefly. A short discussion of the possible pathologic physiology of the deaths will follow. Precautions that may be used to prevent the unfortunate occurrence will then be mentioned. Finally, a few of the nonfatal, but none the less important, complications will be briefly considered.

Hemorrhage

It seems rather strange that in a brief review of the recent literature dealing with the major complications occurring during and after fever therapy, not a single author stressed hemorrhage as a potential danger. Probably the most constant pathologic finding at necropsy of patients who had succumbed after exposure to high temperature has been hemorrhage. I grant that in most cases the hemorrhages are slight and usually petechial, but it would seem that this complication deserves more attention than has been afforded it in the past. As Wilson[2] has pointed out, consideration of the pathologic conditions in cases of fatal heat stroke has apparently been decidedly secondary to consideration of the clinical manifestation, and the precipitating cause of the rapid and uncontrollable rise in temperature and the immediate cause of death are poorly understood. Wilson reported 3 deaths which followed fever therapy and a fourth which followed exposure to the sun. In all of these cases there was extensive hemorrhage under the endocardium of the left ventricle, which was especially severe on the septal wall in the region of the bundle of His. It was suggested that this was probably the actual fatal mechanism, although the cerebral edema and petechial hemorrhages throughout the brain, also present in all of the cases, probably accounted for the early clinical picture of the tremendous rise of body temperature accompanied by unconsciousness. And although Wilson stressed the pathologic conditions present in the heart, it is the presence of hemorrhage that seems most striking.

Five fatalities have been reported by Hartman and Major[3] and by Wilbur and Stevens.[4] The findings at necropsy in all of these cases included hepatic necrosis, hemorrhagic pneumonia, hemorrhagic encephalitis involving the vessels at the base of the brain, subconjunctival hemorrhages, submucosal hemorrhages in the trachea and subendocardial and myocardial hemorrhages.

Chunn and Kirkpatrick[5] reported a case of death after fever therapy in which, in addition to many petechial hemorrhages present throughout the various vital organs, there was a massive hemorrhage into the stomach and intestines. Trautman,[6] discussing the only death which occurred in his group of 1,200 patients who received 6,881 fever treatments, reported, among other findings, considerable congestion of the small blood vessels of the basal ganglia, with hemorrhages in various regions; widespread petechial hemorrhages throughout the cerebellum, and acute congestion and hemorrhage in the spleen. Parenchymatous degeneration of the liver, in various degrees of severity, also has been present in the majority of the cases, frequently together with degeneration of the adrenal glands and hemorrhages into them, as well as various degrees of cerebral edema. Numerous workers, in an attempt to understand better the causes of death from hyperpyrexia, have subjected experimental animals to artificial fever. Widespread petechial hemorrhages and hepatic damage have been reported in every instance.

What, then, is the underlying mechanism of this hemorrhagic tendency accompanying fever? Is there a possible relation between the coexistent hepatic damage and hemorrhages in the fatal cases? Several hypotheses have been advanced as possible answers to these questions.

Wilbur and Stevens expressed the opinion that the hemorrhage might be due to extreme dilatation of the small vessels which allowed extravasation of

the erythrocytes or, possibly, that it was the result of capillary damage not demonstrated anatomically. Rossman[7] has recently reported low levels of capillary resistance, measured by a negative pressure suction test, during artificially induced fever.

Anoxia as a Cause of Hemorrhage. — Hartman[8] noted the great similarity between the pathologic lesions following fever therapy and those due to prolonged asphyxia in acute alcoholism or carbon monoxide or nitrous oxide poisoning. He expressed the belief that anoxia is the damaging factor. He demonstrated that constant and severe anoxia was produced in experimental animals after induced fever.

Wilson and Doan,[9] realizing that prolonged anoxia does result in damage to the hepatic cells, attempted to explain the hemorrhagic tendency during fever therapy chiefly on the basis of hepatic damage, which results in deleterious effects on certain factors important in the coagulation of blood. Prothrombin and fibrinogen, both necessary for the clotting of blood, are produced in the liver, and both have previously been shown to be suppressed in various forms of hepatic damage and after exposure to toxic agents. Hemorrhage has also been shown to occur when the prothrombin level has fallen to less than 40 per cent of the normal value. Consequently, these investigators made extensive studies on the prothrombin and fibrinogen levels and on hepatic function. A series of experimental animals and human subjects was subjected to hyperpyrexia in an effort to see whether there was any appreciable effect on these factors in the range of temperatures commonly used therapeutically. Blood platelet counts and serial biopsies of bone marrow were also done. Apparently no oxygen was administered during the course of the fever sessions.

Microscopic examination of the liver of the experimental animals in this series revealed acute degeneration in each instance. The damage was mild in some animals. However, the levels of prothrombin and fibrinogen were lowered in direct proportion to the extent of destruction of the liver. Each animal also showed scattered local hemorrhages.

Similar observations were made on the human subjects. Pathologic retention of bromsulfalein occurred in each instance, and this was accompanied by an increase of the icteric index. A decrease of the prothrombin level was also noted for each patient studied, the level continuing to fall for several hours after termination of the hyperpyrexia. It is significant that the most marked disturbance of hepatic function occurred in a patient who was not given extra dextrose during the period immediately before fever was induced. In this instance the prothrombin level dropped to 11 per cent of normal on the second day after the fever session, at which time hematemesis occurred. The level had returned to normal by the seventh postfever day. Although the prothrombin in each of the other cases dropped about 50 per cent, it never went below the so-called critical level of 40 per cent, and there was no clinical evidence of hemorrhage. The fibrinogen level was not greatly altered in the cases studied, and this is probably accounted for by a fact pointed out previously, that more extensive hepatic damage is necessary to affect the fibrinogen level than is necessary to lower the prothrombin level. In every instance there was also a fall of the blood platelet level after the fever, following a transitory increase during the actual fever episode. The changing level of the platelets in the peripheral blood reflected directly the state of the megakaryocytes in the bone marrow, there being definite cytoplasmic and nuclear damage during the thrombocytopenic period.

Wilson and Doan concluded from these studies that the pathogenesis of hemorrhage in induced fever may be as follows: the elevation of body temperature results in anoxia and a depletion of glycogen in the liver, which in

turn results in hepatic damage. With sufficient hepatic damage (which is more likely to occur during glycogen deficit) there is a decrease in the concentration of prothrombin, with or without a decrease in that of fibrinogen. There is also direct damage to the megakaryocytes with a resulting decrease of circulating blood platelets. This decrease of platelets, prothrombin and fibrinogen, individually and collectively, contributes to potential or actual hemorrhage. The regeneration of the damaged parenchymatous tissues apparently takes place promptly and completely, the changes being reversible within the usual limits of therapeutic application.

In these cases the major deflections of both the prothrombin level and the platelet counts occurred only after the body temperature had returned to normal, and these values tended to fall for as long as forty-eight hours after the fever sessions, gradually resuming the prefever level about the fifth postfever day. It is interesting to speculate whether or not this prolonged fall possibly could be responsible for heat stroke occurring several hours or days after the termination of hyperpyrexia. This is a common finding in cases of death associated with fever therapy, in which the temperature falls to a normal level as usual after therapy, the patient is apparently in good condition and then suddenly there occurs a rapid and uncontrollable rise of the temperature, usually terminating in death. This condition possibly could be accounted for on the basis of a low concentration of prothrombin, resulting in a hemorrhage into the brain stem in the region of the heat-regulatory center, resultant damage to this center and the ensuing fatal temperature.

The obvious inferences from these observations are that one should do everything possible to prevent this hepatic damage and to take particular care in the selection of cases, so that no one who has demonstrable extensive hepatic damage should be subjected to hyperpyrexia. Also, it might be advisable to give dextrose routinely both by mouth and parenterally before treatment, especially before the longer eight to ten hour sessions, in an effort to build up the glycogen supply of the liver and thus protect this organ as much as possible. In addition, the prevention of anoxia is of prime importance, since hepatic damage *probably will not occur* from heat alone at the therapeutic levels of pyrexia now used in the absence of anoxia. For this reason, oxygen *should be* administered routinely throughout all fever sessions. A few other considerations of this anoxia will now be briefly discussed.

As has already been mentioned, Hartman[8] expressed the opinion that all of the lesions found at necropsy after fever deaths are analogous to those found after death resulting from anoxia from various causes. It has been suggested in an earlier paragraph that anoxia may be the precipitating factor in the hepatic damage frequently observed. In addition, as pointed out by Krusen and Elkins,[10] inferential evidence presented in connection with studies of the results of high fever on the degenerative changes in the peripheral nerves of rabbits indicated that regional anoxia was a contributing factor of considerable importance in the production of these lesions.

Of more immediate practical importance in the average case, however, is the fact that the patient is usually more comfortable while receiving oxygen and his course during the treatment is usually much more stable and more easily controlled. Cullen, Weir and Cook[11] made several comparative observations on three groups of patients undergoing therapeutic hyperpyrexia; one group did not receive oxygen during the treatment; the second group received oxygen therapy only after the desired level of fever had been established, and the remaining group received oxygen with or shortly after initiation of the fever therapy. Striking differences are noted in the clinical courses of the three groups. In the group not receiving any oxygen, the pulse rate climbed rapidly to a high level and continued to increase as the treatment

progressed. In the group given oxygen after the desired level of the fever had been reached, the rate slowed after the oxygen was administered and remained at the lower level during the course of the treatment. In the group given oxygen from the start, the pulse rate became elevated but never reached the high level of the two previous groups, and it persisted at this relatively low level during the course of the hyperpyrexia. In addition restlessness, mental confusion and excitement were much less frequent and less severe among the patients receiving oxygen, and, of greater importance, sedation by drugs was required less frequently, and smaller doses could be used, to control the restlessness. Also, in the absence of oxygen therapy, several treatments had to be discontinued because of either extreme tachycardia of poor quality or uncontrollable excitement, while the same patients tolerated subsequent treatment satisfactorily when oxygen was administered.

What, then, are the factors conducive to this apparent clinical anoxia? There are probably several contributing factors, including the increased temperature of the blood, which, as has been shown, decreases the oxygen saturation, and the increased metabolism occurring with the hyperpyrexia. It has been demonstrated that for every degree rise in temperature that occurs, the basal metabolic rate is increased approximately 7 per cent. This increased metabolism means a great demand for oxygen in the tissues and particularly in the central nervous system, where slight degrees of oxygen deficit are early manifest.

But, important as these two factors may be in relation to the anoxia, Cullen and co-workers felt that probably the most important factor is the alkalosis that occurs soon after pyretotherapy is begun. It will be remembered that under normal conditions the pH of the blood remains remarkably stable at an average level of about 7.4 and with a normal carbon dioxide-combining power of about 60 to 65 volumes per cent. But shortly after the commencement of hyperpyrexia, it has been shown that the rapid, deep breathing incident to higher temperatures results in a blowing off of large quantities of carbon dioxide. The carbon dioxide-combining power thus may be reduced to approximately 40 volumes per cent and the pH of the blood increased to about 7.6. This alkalosis is most important when one considers the need for extra oxygen during fever therapy, since the slightly alkaline hemoglobin compound gives up its oxygen less readily than normal hemoglobin. This results in a significant reduction of the arterial oxygen tension, even in the presence of a normal arterial oxygen saturation. And when it is remembered that the fundamental factor in the supply of oxygen to the tissues is the difference of tension between arterial blood and that of the tissues, it immediately becomes obvious that any interference with a normal oxygen tension of the arterial blood will necessarily reduce the amount of oxygen available to the tissues.

Cullen and co-workers clearly showed this decrease of oxygen tension even in the presence of a practically normal oxygen saturation of the arterial blood during fever therapy. They calculated the percentage of oxygen saturation of the arterial and venous blood before, at the height of and at the completion of artificial fevers of four to five hours' duration at a level of 105 F. At the same time, they measured the pH of the blood, the carbon dioxide content and tension and the oxygen tension of the blood. In addition they noted the effect of the administration of oxygen on these calculations and made the following observations:

1. In the absence of oxygen therapy, the percentage of oxygen saturation of the arterial blood decreased only slightly, even at the height of the fever. The percentage of oxygen saturation of the venous blood, however,

increased rapidly and to a fairly high level. Thus, it was apparent that the oxygen need was probably due, not to a decreased oxygen supply in the blood, but rather either to an interference with the ability of the hemoglobin to make this oxygen available to the tissues or to an interference with the ability of the tissues to utilize the available oxygen.

2. There was a definite increase of the pH of the blood with fever therapy. This increase was apparently directly related to the fall of the carbon dioxide content of the blood, with a resulting decrease of carbon dioxide tension. The carbon dioxide content of the blood had an average drop of 15 per cent in the cases studied.

3. There was a considerable fall of oxygen tension in those cases in which no oxygen was given, and the fall was directly related to the rise of pH and fall of carbon dioxide tension. In these cases, the arterial oxygen tension fell as much as 25 per cent with the development of the desired level of fever and continued to fall as the treatment progressed. This drop in tension was comparable to that obtained by ascent to an elevation of 17,500 feet (5.3 kilometers).

4. With the administration of oxygen by means of the nasal catheter during the entire treatment, there was no drop of oxygen tension, but a slow continued rise, the pH tended to remain at nearly normal levels and there was little decrease of the carbon dioxide tension.

5. When oxygen was administered only after fever had been induced to the desired level, the oxygen tension rose to a point 19 per cent above the original level, after the initial fall, and there was a gradual restoration of pH to near the prefever values.

6. Similar increases of oxygen tension were obtained with the administration of 5 per cent carbon dioxide after the desired level of fever had been reached, but increase above the prefever level was not so great.

From these observations, Cullen and co-workers concluded that the fall of arterial oxygen tension provided a reasonable explanation for the clinical evidence of oxygen want, with a decreased rate of diffusion of oxygen accompanying the fall of arterial tension, and they expressed the opinion that the administration of oxygen benefited the patient chiefly by physical methods. That is, an increase of the alveolar oxygen concentration increases the partial pressure, thereby increasing the saturation of the blood with a consequent increase of oxygen tension, in spite of an elevated pH. The break in the vicious cycle of low tension, high pH and tissue anoxia could also be partially effected by the increased amount of oxygen in physical solution in the plasma, which oxygen is readily available to the tissue and not dependent on dissociation of the hemoglobin.

From these observations, it would seem most advisable to administer oxygen continually throughout the treatment by means of the nasal catheter at a rate of flow of 6 to 8 liters per minute. It is felt that by this means a great deal of the restlessness and excitement can be prevented and that possibly hepatic damage, with the resultant dangers of hemorrhage, can be obviated, as well as all of the other attendant hazards and sequelae of anoxemia.

For example, possibly another direct sequelae of oxygen lack to the brain is cerebral edema. This complication of fever therapy is considered to be fairly common, particularly in those cases in which there are organic diseases of the brain. Its pathogenesis in man is conjectural, but, as has been pointed out by Hartman, cerebral edema in experimental animals always follows anoxia of the brain, with fluid passing through capillary walls at four times the normal rate after only three minutes' lack of oxygen. The same process may well occur in man, and perhaps cerebral edema does occur to a minor degree in many cases and is not recognized as such. Ewalt, Parsons, Warren

and Osborne[12] stated that the earliest recognizable signs of this condition are circumoral pallor, increasing tremor of the lips and fingers and, finally, projectile vomiting. If measures for control are not instituted promptly at this phase, the patient becomes restless, overactive and delirious, this stage being followed by deep coma and at times a severe, generalized clonic convulsion, with a sharp increase in the temperature to 109 F. and death if treatment is not prompt and vigorous. Coincidentally with the first developing signs of this complication, the attack can usually be aborted by the intravenous administration of 100 cc. of 50 per cent solution of dextrose and sucrose. If the patient's condition improves, the hyperpyrexia may be continued cautiously and with a constant watch kept for further danger signs. Oxygen should also be administered, as pointed out previously. If the patient becomes delirious or comatose, the treatment should be terminated. If convulsions develop, spinal drainage should be performed, and if they are not controlled by this measure, sodium amytal or evipal sodium may be given intravenously.

In connection with cerebral edema and cerebral anoxia some interesting observations were made recently by Wood and McCravey.[13] In a series of fever treatments, they noted that several patients had a greatly increased pulse pressure associated with drowsiness and lethargy approaching stupor and that the depth of this stupor was directly proportional to the height of the pulse pressure. These observations were interpreted as signs of cerebral anoxia, which they believed resulted from an increase of volume of cerebrospinal fluid and a decrease of volume of cerebral blood. They expressed the opinion that the increase of volume of spinal fluid was due to an increased meningeal permeability, and that the increased intracranial pressure prevented a normal cerebral blood flow, with resultant cerebral anoxia. With decreased blood flow there occurs a compensating elevation of the systolic blood pressure and, in turn, of the pulse pressure. Stupor in all of their cases was abolished, the pulse pressure assumed a normal level and the patient became more alert after drainage of 24 to 48 cc. of spinal fluid.

It does not seem practical to subject every patient undergoing fever therapy to a spinal drainage if there is a slight increase of pulse pressure, as advocated by these workers. Perhaps routine administration of oxygen would obviate this difficulty. The work does direct proper attention to the value of the pulse pressure, however, as an index for determining the state of the cerebral circulation. Any considerable fluctuation from its normal values demands prompt attention.

Circulatory Collapse

Another serious complication of therapeutic hyperpyrexia is circulatory collapse. This is usually the result of dehydration that may occur as a result of excessive sweating and insufficient intake or absorption of fluids, with a diminution of blood volume and blood electrolytes, especially sodium and chlorides. The symptoms are the same as those observed in circulatory collapse from any other cause, with a rapid, weak, thready pulse and a rapidly falling systolic and diastolic blood pressure. If an intravenous drip of 5 per cent solution of dextrose in physiologic solution of sodium chloride is started with the first suggestions of the complication, the treatment can be continued in many cases, the blood pressure gradually rising and the patient showing general improvement. If the blood pressure continues to fall, however, the treatment should be discontinued at once and the patient gradually cooled. Although recommended by some workers, ephedrine and related drugs should not be used in this condition except as a last resort. Their use will result in a constriction of the peripheral blood vessels with a possible resultant rise of the temperature of the body to dangerously high levels.

Heat Stroke

Heat stroke, previously referred to in connection with fever deaths and intracranial hemorrhage, is another complication of the most serious nature. It is fortunately infrequently encountered. In this condition the patient's temperature may suddenly rise to 108 F. or higher, and despite immediate removal of the patient from the cabinet, may continue to rise. The pulse becomes hard, but its rate is not increased in proportion to the elevation of the temperature. The blood pressure increases, and cerebral and pulmonary edema may suddenly develop. The cause of heat stroke is not definitely known, but apparently there is a derangement of the heat regulating center, possibly due to anoxemia or small hemorrhages about the center. The patient should immediately be removed from the cabinet and cooled by means of a tepid sponge bath and by a fan which blows cool air directly on his body. In this connection, it should be remembered that, as pointed out by Krusen and Elkins, ice packs and ice baths should never be used in an effort to secure a sudden lowering of the body temperature, as the cold contracts the peripheral capillaries and lessens radiation of heat, resulting in an increase of body temperature.

Apprehension and Restlessness

Among the minor complications, which do not greatly jeopardize the patient's life but do interfere with treatment and cause the patient a great deal of discomfort, are restlessness and apprehension. Practically all patients will show a certain degree of these before preceding their first treatment. This is to be expected, and a certain proportion of treatments may have to be discontinued because of marked fear, nervousness and anxiety. This number can be reduced to a minimum, however, if time is taken the day before the initial treatment to acquaint the patient with the procedure to be used, to explain all of the apparatus to him and to tell him that the session may be uncomfortable, especially during the induction phase, but that there is nothing to be alarmed about and that trained nurses and physicians will always be at hand. And, as has been pointed out many times before, a cheerful, skillful technician in charge of the session can do more to gain the cooperation of the patient and allay apprehension during the treatment than any drug that may be used.

Despite reassurance by the physician, however, and a skilful nurse in charge, the individual reaction will vary widely. This may be summarized somewhat as follows:

> Usually the patient's outstanding moods and mannerisms are exaggerated by the hyperpyrexia. For example, the phlegmatic type is little perturbed and is generally cooperative; the aggressive patient commonly manifests signs of aggression and restlessness, while the contented sociable person becomes elated and even euphoric. The hypochondriac is greatly concerned and complains constantly, while instability, if originally present, manifests itself during the hyperpyrexia. Because of these observations, one can often predict the patient's reaction to fever therapy fairly accurately and govern the sedation and management accordingly.

Minor degrees of restlessness while the patient is in the cabinet can usually be controlled easily, either by a word of reassurance or by small doses of sedative drugs. In this conection the choice of the drug used varies widely with different physicians. Probably the most commonly used and safest are the opiates in small doses, particularly pantopon in doses of 1/6 to 1/3 grain (0.01 to 0.02 Gm.) and morphine sulfate in doses of 1/6 to 1/4 grain (0.01 to 0.016 Gm.). Codeine sulfate administered hypodermically in doses of 1 grain (0.065 Gm.) may prove entirely satisfactory. The results gained vary widely with individual patients, however. Possibly an alternation of codeine and pantopon at safe intervals may prove the most effective. Some physicians

stress the efficacy and safety of the carbamides, particularly "sedormid," in doses of 5 to 10 grains (0.3 to 0.65 Gm.), and many like paraldehyde, despite its taste and odor. There seems to be fairly general agreement that the barbiturates should be used cautiously, if at all, during the fever session, as it has been shown by English workers that several of these drugs, particularly phenobarbital and evipal, tend to decrease or abolish utilization of oxygen by the brain, thus contributing to the danger of anoxia. In addition, increased restlessness, approaching delirium, has been noted frequently after their use. Atropine should never be used in combination with any of the other drugs, since sweating may be inhibited and an uncontrollable rise of temperature thus be produced.

When marked restlessness and fear persist despite the foregoing measures, the fever session may have to be discontinued and attempted at another time. Many psychotic patients will have to be restrained while in the cabinet.

Herpes Simplex

Another minor complication, but one that may cause the patient a great deal of pain and discomfort, is herpes simplex. The incidence of this condition varies widely in different parts of the country, some authors reporting its occurrence in 70 per cent of all patients during and after the initial treatment and others reporting an incidence of only 10 to 15 per cent. It is generally agreed that the higher the temperature during the first treatment, the more frequent will be the occurrence of the lesions. Also, patients who have a known tendency toward herpes are more likely to be affected during fever therapy. Its cause is thought by many to be activation of a virus already present in the skin of the face due to the rise of cutaneous temperature during treatment. The lesions occur in the distribution of the facial nerves, with vesicles developing about the nose and mouth and rarely about other orifices.

Because of the possibility that a partial cross immunity exists between the virus of herpes and the vaccine virus, Keddie, Rees and Epstein[14] administered a single smallpox vaccination previous to fever therapy in an attempt to reduce the incidence. There was no reduction of the incidence, however, regardless of the reaction to the vaccine.

Hilton[15] expressed the belief that the painful vesicles are due chiefly to constant local irritation from the wiping away of perspiration by the attending nurse and from cracking and drying of the lips and mucous membranes of the nose from the fever. In his opinion prevention of this trauma and cracking of the mucous membranes will prevent the development of herpes. Accordingly, he removes all sources of local irritation about the nose and mouth and thoroughly oils the skin of the face from the eyes down with olive oil and petrolatum, using care to cover the surface of the lips and the skin on the inside of the nostrils. This is done previous to the induction of the fever and frequently during each treatment, and wiping of the face is reduced to a minimum. Although he did not present any statistics or controls, he stated that this regimen if carefully carried out will prevent herpes simplex. Ewart[12] recommended a trial heating of one hour three to seven days before the first therapeutic dose, and stated that this will reduce the incidence of herpes to less than 0.5 per cent. Once the lesions have developed, the application of compound tincture of benzoin or 50 per cent alcohol and 1 per cent phenol usually hastens the drying of the vesicles and reduces the pain.

In a few rare cases, ocular herpes may develop, and this complication demands prompt attention. The pupil should be dilated with atropine. Instillation of 1 per cent solution of butyn sulfate will help relieve the pain. The following session of hyperpyrexia will usually reduce all inflammation and obviate any danger of perforation or scarring of the cornea.

Nausea and Vomiting

The last complication to be considered briefly is nausea and vomiting. This is a fairly frequent development, some authors reporting its occurrence in 25 to 35 per cent of all treatments, but it is rarely severe or persistent enough to cause the termination of treatment. It is an indication, however, for parenteral administration of fluids in an effort to prevent dehydration and disturbances of the blood electrolytes, and the intravenously administered fluids will nearly always terminate the vomiting. In some cases the vomiting may be due to gastric retention of the fluids taken during the febrile period, since the hyperpyrexia in many cases inhibits normal gastric emptying and absorption of fluids. The resulting abdominal distention should be watched for carefully, since this is a sign that a proper fluid level is not being maintained. The nausea and vomiting may be due to gastric irritation from the ingested saline solution. Eating prior to treatment has been shown to increase their incidence greatly. At any rate, the intravenous use of dextrose and saline solution is usually all that is necessary in the management of this condition.

Summary

A few of the complications of artificially induced fever have been discussed briefly. The potential and actual dangers of hemorrhage have been pointed out, the need for the administration of oxygen during the fever stage has been emphasized and the possible need for extra dextrose before treatment has been suggested. Despite its widespread use with a low mortality rate, the fact should always be remembered that therapeutic hyperpyrexia is a potentially dangerous procedure. And the useful axiom should always be recalled: When in doubt, take the patient out of the cabinet.

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DIATHERMY IN PROTRACTED (UNRESOLVED) PNEUMONIA *

EVERILL W. FOWLKS, M.D.

United States Veterans Administration

PORTLAND, OREGON

Diathermy is one of the most valuable single agents in the entire therapeutic armamentarium owing to the fact that heat is developed more or less centrally within the tissues themselves and diathermy is not dependent on an effort to drive heat through the skin and the subcutaneous tissues by application from without.

American physicians are accredited with the first actual use of diathermy in the treatment of lobar pneumonia. In 1916 Price[1] wrote about its application. Sampson[2] in 1923 contributed an article to this field, and De Kraft[3] added to the knowledge of its use in 1922.

Stewart[4] wrote an entire textbook on its use in 1923 and revised it in 1926. Several officers of the United States Public Health Service have contributed articles and reports of what they have observed in the employment of diathermy in the various Marine Corps hospitals.

In reviewing the articles written on this subject, one discovers that all represent observations which were poorly controlled. The general conclusions in these reports all point toward the favorable outcome of the disease, but there appears to be uncertainty as to any specific action or reaction caused by the employment of diathermy. Stewart,[4] Harvey,[5] Jenkins,[6] Robinson[7] and Wetherbee[8] all felt that the mortality rate was decreased in their cases but the morbidity unaltered. Simon[9] of St. Bartholomew's Hospital in London, failed to show any decrease in the mortality rate in his series.

The ushering in of the "sulfa" era of medicine has caused a steady decrease in the use of diathermy in pneumonia as well as in pulmonary abscess. The use of the specific serums, however, for some unknown reason, did not check the use of diathermy as much as the sulfonamide compounds. In the literature one is able to find numerous reports of the combined use of serum therapy and diathermy with very encouraging final results.

Recently, at the Portland United States Veterans Administration Facility, I reviewed the cases of pneumonia observed at the hospital in the previous six years. Many of these cases, of which there were about 400, had come under my direct observation, and many I had observed indirectly. The various sulfonamide compounds had been employed, as well as specific serums when indicated. As soon as a diagnosis of pneumococcic infection was made, an attempt was made to determine the type of Diplococcus pneumoniae responsible. This is difficult after administration of a sulfonamide compound has been started. A blood culture proved of value in cases in which the toxemia was severe. In most of these cases the infection was due to pneumoniae of type III or type VII and it was imperative that both serum therapy and chemotherapy be employed.

It is not the plan of this paper to consider in detail the therapy and the clinical and physical findings in cases of pneumonia, but rather to consider one of its most common complications, namely, lack of resolution. During

* This work was done by Dr. Fowlks under the direction of Dr. F. H. Krusen, while he was on assignment at the Mayo Foundation for instruction in physical medicine.

the winter season of 1939-1940, I became very much interested in this phase of pneumonia and started to search the local literature for hints on a more thorough treatment and knowledge of it. It soon became apparent that little attention had been paid to this complication in the literature and still less to the use of diathermy in treating it. Bierman[5] stated that he felt that diathermy should be considered as a possible aid but failed to enlarge on this view.

At our station we have observed that the number of cases of delayed resolution has definitely increased since the employment of the various sulfonamide drugs. The increase has been estimated to be between 22 and 24 per cent. We found that delayed resolution occurred in 40 per cent of all cases in which these drugs were employed. After some bad results followed the early dismissal of patients from the hospital, we discovered that in more than half of such cases roentgenograms revealed a definite density indicative of unresolved pneumonia even after the temperature had returned to normal and the physical and clinical findings were considered to be within normal range. Again I searched the medical literature for an answer to this problem and was unable to discover any article which adequately explained why delayed resolution should follow the administration of sulfapyridine, sulfathiazole or sulfadiazine. It is my opinion that employment of the sulfonamide compounds in the treatment of pneumonia upsets the normal pattern of temperature reduction (lysis and crisis) and that the new pattern created by their action does not keep the temperature high for a sufficient length of time to bring about thermic death of D. pneumoniae by the method nature has provided as a line of defense against infection. I also believe that the normal leukocytic and phagocytic responses are checked or reduced early by the sulfonamide compounds and that a slow state of resolution is thus produced, owing to the reduced amount of natural ferments. In support of these views I offer the fact that repeated blood counts in cases of pneumonia in which the sulfonamide compounds have been administered show an early and constant reduction of the leukocyte count from that determined when the patient was first admitted to the hospital. This reduction is striking when the blood counts are compared with those taken before the sulfonamide compound era. Likewise, the tendency toward secondary anemia is increased.

Using these theories as a working basis, I started to employ diathermy to produce fever in cases of delayed resolution. In all cases, however, a trial of intensive iodide therapy was first carried out. If after ten days, or two weeks at the longest, I discovered that the patient was not progressing satisfactorily from both a physical and a clinical standpoint, the iodides were supplemented with the application of diathermy to the region of unresolved pneumonia. At our station we have used a pancake coil over the thorax for thirty minutes each day until either a definite degree of improvement was noted or a stationary condition was found to exist.

In about 8 per cent of the cases in which this type of therapy was employed, no improvement was observed. It has been the policy at our station to treat these patients with high voltage roentgen therapy provided no contraindications exist. We feel that about 32 per cent of our patients have received some benefit from the employment of diathermy.

Diathermy has not, however, proved to be of any benefit in the prevention of empyema, pulmonary abscess or cardiac complications. It does appear to cause a slight retardation of the pulse rate, a slight lowering of the respiratory rate during and after treatment and a definite reduction of the amount of pleuritic pain in all cases. The blood pressure, both systolic and

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diastolic, fell slightly in all cases in which it was checked. Perspiration was always increased both locally and generally. The amount of sedation required was definitely reduced, and the interval between doses was increased. Sleep was restful and prolonged. In cases in which a low grade fever continued, there was a noticable abatement of the elevation of temperature. The exudate apparently was absorbed faster, as pointed out by Bierman.[10]

In conclusion I wish to bring out the fact that the use of diathermy in protracted pneumonia is thought to be worth while, but that, like all other forms of therapy, it has its shortcomings and failures. In some of the cases in our series, empyema, pulmonary abscess, chronic bronchitis and bronchiectasis have developed. In other cases, patchy areas of pneumonic consolidation have developed. In several of these cases surgical intervention finally has been required. The mortality rate in this series averaged 8.2 per cent, which was considered by us to be a good figure in view of the number of chronic alcoholics, the cases of terminal pneumonia and the number of elderly and middle-aged patients.

Finally, I wish to point out that this is an excellent field for research, as the literature on the subject is conspicuous by its absence.

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ATTENDANCE AT WASHINGTON SESSION APRIL 8TH

Please notify Secretary as early as possible of your intention to be present to aid in planning local eating and transportation problems. Special directions will be forwarded on request. G. J. P. Barger, M.D., Secretary, Eastern Section, American Congress of Physical Therapy, 1125 Buchanan Street, N. W., Washington 11, D. C.

ARCHIVES of PHYSICAL THERAPY
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EDITORIALS

ADVANCES IN NEUROMUSCULAR PHYSIOLOGY

Knowledge of muscular activity in terms of neuromuscular physiology in health and disease is of prime importance as it forms a basis for correct diagnosis and treatment. Such information is of particular value in relation to Physical Medicine for the study of electrical activity of nerves and muscles is often an important aspect of diagnosis, and physical methods of treatment should be employed in relation to their effects on physiologic processes. Recent publications from a number of research laboratories have brought important additions to our knowledge of muscular function which should be incorporated in physical medicine practice.

Paul Weiss[1] has for several years studied nerve regeneration and has concluded that regenerating axons do not reach their appropriate end organs because of chemical attraction from the periphery, but advance along surfaces or interfaces, are guided by the molecular or micellar orientation of these interfaces along which they extend, and do not branch unless obstructed. Optimum return or function would seem best achieved when scar is eliminated by sutureless splicing and the outgrowing fibers proceed along the old pathways to the former motor end plates (in case of motor nerves). The work of Young[2,3] indicates that if muscular atrophy is severe new end plates must be formed and recovery may never be complete. Measures aimed at lessening the degree of atrophy and improving the blood supply therefore favor optimum regeneration. This accordingly is a basis for physical therapy in peripheral nerve injuries including the use of heat, massage, exercise and electrical stimulation. Even after regeneration coordinated movements may fail to return as shown in animal experiments and in clinical material[4,5] as muscles with antagonistic action may contract simultaneously. This may be due to random regeneration of fibers and muscle reeducation is necessary to improve motor function.

By means of microelectrodes and high speed recording technics Lloyd[6] has collected new facts concerning the functional organization of the spinal cord. He has shown that the myotatic stretch reflex is mediated through arcs of two neurons and appears only in the muscle or part of a muscle subjected to stretch. The flexor reflex, however, following stimulation of smaller caliber cutaneous or muscle nerve fibers, has the attributes of a multi-neuron-arc discharge. The interneurons intercalated in the minimum reflex pathway provides diffusion to many synergically acting muscles. Internuncial neurons of the motoneuron pool were found to be inhibited by pyramidal tract discharges in some experiments and reflex activity over two neuron arcs was inhibited by the action of multineuron arcs. This internuncial premotor system may possibly provide the mechanism of reciprocal innervation. Further elucidation of the intricacies of spinal cord activity will help to explain the clinical findings of muscular paralysis, spasm and incoordination, and the significance of the histopathologic changes in the internuncial cells, as described in poliomyelitis recently.[7]

Change in electrical excitability of muscle is one of the well-known effects of motor denervation and testing the responses of muscles to galvanic

and tetanizing (faradic) currents for the reaction of degeneration of Erb is familiar to all physical therapists. More exact quantitative measurements are made by the determination of the chronaxie value or strength duration curves. Progressive currents of long duration may also be used for purposes of electrodiagnosis as well as for treatment.[8] Such quantitative measurements are useful in evaluating peripheral nerve injuries.[9]

The development of sensitive oscillographs capable of recording minute electrical discharges from muscle and nerve has opened up an interesting field of investigation. Recently spontaneous discharges from resting muscles have been found to have characteristic appearances such that complete denervation can be differentiated from beginning reinnervation by regenerating fibers.[10-11] The processes of recovery may be studied by this means and diagnosis as to the degree of injury aided. Recording of action potentials has been found of great value in studying muscular behavior in poliomyelitis and other neurological conditions, and also as a method of evaluating the effectiveness of physical therapeutic procedures.[12-5]

It is now generally accepted that acetylcholine is concerned in the transmission of motor nerve impulses. Carey[13] has shown that the motor end plate may expand to twice the normal size during muscular activity and afford large surface areas for transmission of the nerve impulse, this finding being consistent with the humoral theory of transmission. Nachmansohn[14-15] working with the enzyme choline esterase has shown that acetylcholine is metabolized at a rate compatible with the speed of transmission of impulses across the neuromyal junction and that the energy provided by the chemical reactions are sufficient to account for the electrical potential discharged. Thus the electrical and chemical theories of transmission are brought closer together.

The advances in our understanding of the chemistry of muscular contraction have been ably reviewed by Harpuder.[16] The problem of muscle fatigue which is of such interest in connection with the training of troops has also been summarized. There are two separate mechanisms: one a chemical fatigue of the contracting muscle fibers, and a transmission fatigue of the motor end plates. The clinical significance of the latter is still unknown. As electrolytes such as potassium and other drugs including prostigmine have important effects on transmission and fatigue, they have already been used in treatment of neuromuscular disorders in conjunction with physical therapy, although their effectiveness is not yet fully established.[17-18]

These continual additions to our knowledge of structure and function of the neuromuscular system form the basis for scientific Physical Medicine and open avenues for further researches.

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EDUCATION AND PHYSICAL MEDICINE

It becomes more and more apparent that physical medicine is coming at last into its own and it is now safe to predict that our universities and medical schools are about to give substantial recognition to this highly important field of medical endeavor. The significant role of physical medicine in modern medical education was brought home forcibly at the annual Congress on Medical Education and Licensure held in Chicago, Illinois, on February 14, 1944.

The first two papers presented at this Congress both stressed the importance of physical medicine in modern medical education. In his opening address, Dr. Ray Lyman Wilbur, the Chairman of the Council on Medical Education and Hospitals, said in part, "We are beginning to see how environment and changes have followed us through our evolutionary rise out of the sea. . . Medicine based on pills and potions is becoming obsolete. . . . The last war is said to have put orthopedic surgery on its feet. This war may well do the same for physical medicine. Those treatments involving the use of heat, cold, water, electricity, movement and massage have striking biological responses, including effects upon psychic reactions more potent than many of the drugs gathered through many centuries by trial and error. . . . Nurses, laboratory workers, physical therapists, technical assistants, secretaries and pharmacists multiply what the physician can do for his patient and for the public. . . . The way we use the hospitals and medical schools of today will largely determine the medical future of our people. . . . At this time military medicine, emergency surgery, the relationship of medicine to society and physical medicine should have special emphasis."

Dr. Wilbur left no doubt whatever in the minds of his listeners concerning his belief that physical medicine requires special attention from our medical educators at this time.

Immediately after Dr. Wilbur's address, Dr. Harold S. Diehl, Dean of the Medical Sciences of the University of Minnesota, said: "In most medical schools, the curriculum has become overcrowded and rigid; yet new developments and even some new fields, such as physical medicine and social medicine, must be included in the instructional program if our graduates are to be prepared to deal with the medical problems of the future. In one of the large clinics of this country, 10 per cent of all patients are referred to the division of physical medicine; a field in which few medical schools provide any instruction worthy of the name."

Your editor was, of course, delighted to find that these distinguished medical educators were thus advocating the teaching of physical medicine before the most important annual congress on medical education. There is little room to doubt that, when such outstanding leaders in medical education stress the importance of teaching of physical medicine, it will be developed by the deans of medical schools who heard these men speak. Obviously we are facing a new era in the teaching of physical medicine in our medical schools. Truly the future of physical medicine looks exceedingly bright.

PHYSICAL MEDICINE CENTER

The future development of Physical Medicine as a recognized medical specialty is dependent on a sound scientific basis for the employment of physical procedures in the diagnosis and treatment of disease and injury and more extensive training of talented investigators and clinicians in this field. Properly controlled and coordinated basic and clinical research can best be achieved through the combined efforts of laboratory workers and clinicians as found in university medical centers especially when combined endeavors are correlated under capable directors. The training of physical medicine specialists can also be ideal in such a proposed setup. One of the greatest immediate needs in the field of Physical Medicine has been found to be that of securing an adequate number of teachers in this subject, according to the recent survey of the Baruch Committee on Physical Medicine. The establishment of a Physical Medicine Center at the University of Pennsylvania through the aid of the National Foundation for Infantile Paralysis is a step forward in meeting this great need. Under the skillful guidance of Doctor Peirsol one will look to this center for important fundamental advances in Physical Medicine in the future. It is hoped that similar new centers of such an ideal character may be established.

WAR-TIME GRADUATE MEDICAL MEETINGS

The educational program for the benefit of the physicians in the armed services and civilians under the auspices of the American Medical Association, the American College of Surgeons and the American College of Physicians is now well under way and reports indicate that the clinics and lectures which have been given at numerous military centers have been quite successful. Although only a few hours can be devoted to Physical Medicine at any one location, because of the nation-wide scope of these meetings it is possible to inform a considerable number of physicians of the fundamental principles of physical therapeutics and some of the important benefits to be obtained especially in relation to rehabilitation. In order to aid the lecturers in Physical Medicine the Council on Physical Therapy of the American Medical Association has prepared a collection of slides which can be obtained on loan by writing Mr. Howard A. Carter, Secretary of the Council at 535

North Dearborn Street, Chicago, Illinois. These ninety-eight slides should be of great aid to the lecturers for they illustrate some of the known physiologic effects of physical agents and the technic of administration of local heat and cold, general heat, diathermy, ultraviolet, galvanic and faradic currents, hydrotherapeutic procedures, massage, therapeutic exercises, certain types of functional Occupational Therapy, and mechanical devices for splinting and exercising. It is important for the advancement of Physical Medicine that we all cooperate to the fullest extent in making these war-time graduate medical meetings interesting and instructive.

AMERICAN HOSPITAL ASSOCIATION RECOGNIZES NEED FOR PHYSICIANS TRAINED IN PHYSICAL MEDICINE

There is unquestionably a great shortage of well-qualified physicians who can specialize in the field of physical medicine. We understand that the Baruch Committee on Physical Medicine has found this need for qualified physicians who can specialize in physical medicine to be paramount. The Board of Trustees of the American Hospital Association has also recognized this need and at its meeting on December 4, 1943, this board prepared a resolution which read in part as follows: "The Board of Trustees recognize, in view of the dearth of well-trained medical men who are specializing in physical therapy, that there be considered ways of encouraging the training of a larger group of medical men in this specialty."

It becomes apparent that one of the first things to be done in order to bring physical medicine into its own in this country is to provide means for the training of qualified young physicians to specialize in our rapidly developing branch of medical practice.

Albert Franklin Tyler 1881-1944

Just as we are going to press we learn with deep regret of the death of Dr. Tyler one of the founders and past-presidents of the Congress. An obituary will appear in the April issue.

MEDICAL NEWS

Subcommittee Members Baruch Committee on Physical Therapy

Subcommittee on Poliomyelitis

Ray Lyman Wilbur, M.D., *Chairman,*
Standford University, California.
Donald Gudakunst, M. D.,
New York, N. Y.
Frank H. Krusen, M.D.,
Rochester, Minnesota.
Mr. Basil O'Connor,
New York, N. Y.

Subcommittee on Public Relations

Ray Lyman Wilbur, M.D., *Chairman,*
Stanford University, California.
John S. Coulter, M.D.,
Chicago, Illinois.
Morris Fishbein, M.D.,
Chicago, Illinois.
Mr. Lawrence C. Salter,
Chicago, Illinois.

Subcommittee on Occupational Therapy

Kristian G. Hansson, M.D., *Chairman,*
New York, N. Y.
Mr. Everett S. Elwood,
Philadelphia, Penna.
Robert W. Johnson, Jr., M.D.,
Baltimore, Maryland.
Winfred Overholser, M.D.,
Washington, D. C.

Subcommittee on Prevention

Kristian G. Hansson, M.D., *Chairman,*
New York, N. Y.
Lloyd T. Brown, M.D.,
Boston, Massachusetts.
Frank Ewerhardt, M.D.,
St. Louis, Missorui.
Royal S. Haynes, M.D.,
New York, N. Y.
William B. Snow, M.D.,
New York, N. Y.

Subcommittee on Hydrology and Health Resorts

Carl R. Comstock, M.D., *Chairman,*
Saratoga Springs, N. Y.
Robert Lee Bennett, M.D.
Warm Springs, Georgia.
Victor Diederich, M.D.,
Hot Springs National Park, Arkansas.

Subcommittee on Clinical Research

John S. Coulter, M.D., *Chairman,*
Chicago, Illinois.
Frank D. Dickson, M.D.,
Kansas City, Kansas.
George M. Piersol, M.D.,
Philadelphia, Penna.
Arthur L. Watkins, M.D.
Boston, Massachusetts.
Miss Catherine Worthingham,
Palo Alto, California.

Subcommittee on Rehabilitation

Capt. Charles F. Behrens (MC), U. S. N., *Chairman,*
Bethesda, Maryland.
Lt. Col. Benjamin A. Strickland, Jr. (MC), U. S. A., *Vice-Chairman,*
Tucson, Arizona.
Major Walter E. Barton (MC), U. S. A.
Washington, D. C.
Ensign Jane Cassedy,
Bethesda, Maryland.
Lt. Glenn Drewyer (MC), U. S. N. R.,
Glenwood Springs, Colorado.
Lt. Comdr. Harry Etter (MC), U. S. N.,
Bethesda, Maryland.
Major Ora Huddleston, A. U. S.,
Denver, Colorado.
Lt. Comdr. Robert Kinneman (MC), U. S. N. R.,
Great Lakes, Illinois.
Capt. Howard Montgomery (MC), U. S. N.,
Washington, D. C.
Lt. Col. Howard A. Rusk (MC), U. S. A.,
Washington, D. C.

Subcommittee on Basic Research

William T. Sanger, M.D., *Chairman,*
Richmond, Virginia.
Detlev W. Bronk, Ph.D.
Philadelphia, Penna.
Alan Gregg, M.D.,
New York, N. Y.
Andrew C. Ivy, M.D.,
Chicago, Illinois.
Eugene Landis, M.D.,
Boston, Massachusetts.

Subcommittee on Teaching

Frank H. Krusen, M.D., *Chairman,*
Rochester, Minnesota.
Robin Buerki, M.D.,
Philadelphia, Penna.
Mr. Howard Carter,
Chicago, Illinois.
Frances A. Hellebrandt, M.D.,
Madison, Wisconsin.
Ernest Jaqua, Ph.D.,
Washington, D. C.

War Production Board Order L-259

The War Production Board is presently considering a revision of General Limitation Order L-259 (Physical Therapy Equipment) to provide for the manufacture of ultraviolet radiation equipment, in-

fra-red lamps, and electric bakers for sale to the following in addition to the Army, Navy, Maritime Commission, War Shipping Administration and Lend-Lease:

1. Hospitals and medical departments of industrial concerns.

2. Licensed medical practitioners ("Licensed medical practitioner" means any person located in the United States or Canada who is licensed by the competent legal authority to practice medicine or any of the healing arts and whose license permits him to use physical therapy equipment in his practice).

3. Any other person who presents a written order signed by a licensed medical practitioner calling for this equipment.

4. Holders of export licenses.

Soon after Order L-259 restricting the manufacture and sale of physical therapy equipment was issued by the War Production Board, Dr. John S. Coulter, Chairman of the Council on Physical Therapy of the American Medical Association, and other members of the Council began an effort to have the Order liberalized. A group of the manufacturers of physical therapy equipment, who too felt that the Order was unnecessarily restrictive, urged the War Production Board to liberalize the Order and tried to cooperate with the undertaking of the physicians in every way that was proper and practical.

The general attitude of the physicians and the manufacturers was that there was no desire to interfere in any way with the war effort but that if there were any material over and above that required in the prosecution of the war they believed physical therapy equipment should have consideration on account of its value in restoring to health and useful war effort those who were ill and injured.

Dr. Coulter made several trips to Washington and elsewhere, exerted great effort and had great influence in convincing the War Production Board and its advisors that Order L-259 could wisely be liberalized. Unfortunately, in the midst of these efforts on the part of Dr. Coulter, he became ill and for many months was forced on this account to forego all of his many activities, including this one. Dr. Coulter asked Dr. Frank H. Krusen, a member of the Council on Physical Therapy, to carry on the endeavor in behalf of the physicians using physical therapy equipment and the manufacturers serving them. Dr. Krusen has made several trips to Washington and has done an outstanding job in the capacity of advisor to the War Production Board. He has had much to do with the entire matter and particularly the final drafting of a very liberal proposed Order.

It appears that due to the good work of Dr. Coulter and Dr. Krusen, Order L-259 will be revised in the early future and will be liberalized to the extent that we can resume the manufacture and sale of physical therapy equipment on a basis adequately liberal for present day needs. Exactly when this revised Order will be promulgated is, of course, unknown to us. It is our opinion, putting together all the information obtainable from different sources, that the revision will be coming through in the early future—perhaps within a month.

It is our belief that physical therapy equipment will be permitted to be manufactured for sale to all "licensed medical practitioners."

Both Dr. John S. Coulter and Dr. Frank H. Krusen, in our opinion, deserve great credit for lending their weighty and effective influence and expending much effort on behalf of the physicians using physical therapy equipment and the manufacturers who serve these physicians.

Dr. Piersol Heads New Program in Physical Medicine

Dr. George M. Piersol, professor of medicine in the Medico-Chirurgical College, Graduate School of Medicine, University of Pennsylvania, and a member of the Council on Physical Therapy of the American Medical Association, has been appointed director of the new Center for Research and Instruction in Physical Medicine in the graduate school of medicine of the university. The center was established by the National Foundation for Infantile Paralysis when a grant totaling $150,000 was given for a five year period from Jan. 1, 1944 to Dec. 31, 1948. Dr. Piersol will relinquish his private practice to direct the center, one of whose objectives is to explore thoroughly the possibilities of physical means of treatment not only of infantile paralysis but of other diseases as well. Dr. Piersol graduated at the University of Pennsylvania School of Medicine in 1905, becoming a member of the faculty in 1907.

New York Physical Therapy Society

The regular March meeting of the New York Physical Therapy Society was held March 1, at the New York Academy of Medicine Building. The program was as follows: "Some Physical Therapy Considerations in Poliomyelitis in View of the Kenny Concept," by William Bierman, M.D., and Ruth Bergman, Jr. R. P. T. T. The discussion was opened by John L. Lavan, M.D., D.P.H.; Joseph Moldaver, M.D., and Philip M. Stimson. An executive session followed.

Drs. Hansson and Krusen Guest Speakers at Physical Therapy Meeting

On Feb. 4 at the Hotel Pennsylvania in New York City the New York Chapter of the National Foundation for Infantile Paralysis, Inc., met jointly with the New York and Pennsylvania Chapters of the American Physiotherapy Association. The guest speakers were Dr. K. G. Hansson, President of the American Congress of Physical Therapy, Dr. Frank H. Krusen and Mr. Basil O'Connor.

Associate Dean Named to Emory

Dr. Robert Morris Paty of the Bibb Manufacturing Company, Porterdale, has been appointed associate dean at Emory University School of Medicine and medical director of Emory University Hospital, Atlanta.

Dr. Kovács in Demand as Speaker on Physical Therapy

Dr. Richard Kovács, Secretary of the American Congress of Physical Therapy, is much in demand as a speaker on physical therapy, as evidenced by the following:

At the Columbus Hospital, New York, Dec. 14 he spoke on "Recent Progress in Physical Therapy." During the week of Jan. 24 he addressed the Richmond, Va., Academy of Medicine on physical therapy. Under the auspices of the New England Committee on Wartime Postgraduate Medical Meetings he presided at a symposium on physical therapy Feb. 17 at the U. S. Naval Construction Center Dispensary, Davisville, R. I. He is scheduled to speak on physical therapy in Jacksonville, Fla., March 26 and in Pensacola, Fla., April 1.

Twenty-One War Sessions to Be Held by American College of Surgeons

The schedule for the meetings is as follows:

Date	City	States and Provinces	Headquarters
Feb. 28	Winnipeg	Manitoba, Saskatchewan	The Fort Garry
Mar. 2	Minneapolis	Minnesota, North Dakota, S. Dakota	Hotel Nicollet
Mar. 4	Des Moines	Iowa, Eastern Nebraska, Missouri	Hotel Fort Des Moines
Mar. 6	Chicago	Illinois, Wisconsin	The Stevens
Mar. 8	Cincinnati	Ohio, Kentucky, Indiana, West Virginia, Tennessee	Netherlands Plaza
Mar. 10	Detroit	Michigan	Hotel Statler
Mar. 13	Rochester	New York State	Seneca Hotel
Mar. 15	Toronto	Ontario	Royal York Hotel
Mar. 17	Montreal	Quebec, New Brunswick, Nova Scotia, Pr. Edward Island, Newfoundland	Mt. Royal Hotel
Mar. 20	Springfield	Massachusetts, Maine, New Hampshire, Vermont, Rhode Island, Connecticut	Hotel Kimball
Mar. 22	Philadelphia	Pennsylvania, New Jersey, Delaware	The Bellevue-Stratford
Mar. 24	Baltimore	Maryland, District of Columbia, Virginia, North Carolina	Lord Baltimore Hotel
Mar. 27	Jacksonville	Florida, Georgia, Alabama, S. Carolina	The George Washington
Mar. 31	San Antonio	Texas, Louisiana, Mississippi, New Mexico, Mexico	The Gunter Hotel
Apr. 4	Tulsa	Oklahoma, Kansas, Arkansas	The Mayo
Apr. 7	Denver	Colorado, Wyoming, Western Nebraska	Shirley-Savoy Hotel
Apr. 11	Salt Lake C	Utah, Southern Idaho	Hotel Utah
Apr. 14	Spokane	Washington, North'n Idaho, Oregon, Montana	Davenport Hotel
Apr. 18	Vancouver	British Columbia, Alberta	Hotel Vancouver
Apr. 24	San Fran'co	Northern California, Nevada	Hotel Mark Hopkins
Apr. 27	Los Angeles	Southern California, Arizona	The Biltmore Hotel

Dr. Lowry Speaks on War-Time Program

Dr. Franklin P. Lowry will lecture on Physical Therapy as a part of the War-Time Graduate Medical Meeting scheduled for March 16, 1944 at the U. S. Naval Air Station, Brunswick, Maine.

Dr. Moor Gives Lecture on Fever Therapy

Fred B. Moor, M.D., Professor of Pharmacology and Therapeutics in the Medical School of the College of Medical Evangelists and Director of the School of Physical Therapy, delivered a lecture on "Fever Therapy in the Venereal Diseases" at the Physicians Post Graduate Venereal Disease Training Program at the University of Southern California on Jan. 6.

The Kretschmer Lecture

Dr. Russell L. Haden, Cleveland Clinic, Cleveland, will deliver the third Edwin R. Kretschmer Memorial Lecture on April 28 at the Palmer House. His subject will be "The Varying Clinical Picture of Leukemia."

Lieut. Strance Directs Physical Therapy

The 34th General Hospital is one of the three general hospitals in operation at present exclusively for the California-Arizona Maneuver Area troops. Col. Walter L. Richards, M. C., is the commanding officer. Lieut. John G. Strance, M. C., is the Medical Officer in charge of physical therapy.

Warning on Mineral Water to Relieve Rheumatism

A report has been received that orders are being taken by the Rutherford Laboratories, Post Office Box 143, Westwood, N. J., for "Canada Mineral Water," which, it is said, is being sold to relieve rheumatism. One check for $5 was cashed by "Dr. Henzling." Inquiries directed to the laboratories are said to bring no response. The Bureau of Investigation of the American Medical Association does not have a record of the Rutherford Laboratories or of "Canada Mineral Water." Neither is it able to identify the "Dr. Henzling" reputed to be connected with the firm.

Bill for the Rehabilitation of the Disabled

A disabled persons employment bill, introduced by the government, has received a warm welcome in the House of Commons. For the first time, rehabilitation and resettlement are treated as a single problem, combining medical science and industrial knowledge.

Dr. Magee Speaks on Tropical Medicine

Dr. James C. Magee, Major General, U. S. A. (retired), former Surgeon General and newly appointed Director of Medical Informational Service of the National Research Council, addressed a special meeting of the staff and students of the University of Texas School of Medicine on the significance of tropical diseases before and after the war.

Medical Symposium

One of the interesting papers as a part of the medical symposium held in Durham, N. C., under the auspices of the staff of Watts Hospital was presented by Dr. Tinsley R. Harrison, Winston-Salem on the subject of "The Abuse of Rest as a Therapeutic Measure in Patients with Cardiac Disease."

Dr. Watkins on Wartime Postgraduate Medical Meeting Program

The New England Committee on Wartime Postgraduate Medical Meetings held a symposium on physical therapy at the Portsmouth Naval Hospital Jan. 20. Dr. Watkins presented the subject, "Some Principles of Physical Medicine as Applied to Traumatic Conditions, Particularly the Diagnosis and Treatment of Nerve Injuries."

Dr. Madge C. L. McGuinness Chairman of Special Committee on Physical Therapy

The Medical Society of the County of New York has had a committee on physical therapy since 1926. The Chairman of this committee for 1944 is Dr. Madge C. L. McGuinness. Dr. A. Bern Hirsh is one of the members of the committee.

Meeting of the Committee on Post-War Medical Service

The Committee on Postwar Medical Service met in Washington, D. C., on Jan. 14, 1944. There were present:

Dr. Roger I. Lee, Chairman

Dr. Arthur W. Allen	Dr. W. F. Ossenfort (U. S. P. H. S.)
Dr. Francis G. Blake	Dr. W. W. Palmer
Commander Edward L. Bortz (Navy)	Dr. James E. Paullin
Dr. Fred A. Coller	Dr. George Morris Piersol
Captain W. E. Eaton (Navy)	Brigadier General Fred Rankin (Army)
Dr. Morris Fishbein	Dr. H. H. Shoulders
Dr. Alan Gregg	
Dr. Charles M. Griffith (Veterans Bureau)	

Medical Science Center for Detroit

Under the auspices of the board of directors of Wayne University in Detroit a project is now under way to create a $50,000,000 Medical Science Center to be built around Wayne University's College of Medicine.

List of Slides for Physical Therapy Graduate Medical Meetings

The following slides can be obtained on a loan basis by application to the Secretary of the Council on Physical Therapy of the American Medical Association, 535 North Dearborn Street, Chicago 10, Ill.

Those available are listed as follows:

Thermotherapy: Local Heat

Slide 1: Physiologic effects of cold, heat and exercise. A study of capillaries illustrating the physiologic changes in blood vessels due to exercise and changes in environmental temperatures. Heat should be prescribed. As is shown from this study there is a distinct difference in the effects of mild and marked heat.

Slide 2: Effects of heat on the capillary nail bed under a microscope. Note that heat increases the number of capillaries and capillaries are only visible when they are filled with blood.

Slide 3: Effects of heat on the lymphatic circulation. In this experiment a dye injected under the skin is absorbed by the lymphatics. In the column under control arm, after five minutes, there is very little absorption while in the heated arm there is considerable absorption shown by the spreading of the dye in the capillaries. (Reference: McMaster, P. D.: Changes in the Cutaneous Lymphatics of Human Beings and in the Lymph Flow Under Normal and Pathological Conditions, Jour. Exp. Med., Vol. 65, 1937, p. 347.)

Slide 4: A simple method of applying local heat by means of an inexpensive luminous heat lamp with clamp attachment.

Slide 5: Home made baker. This baker is designed for applying heat to the legs or arms.

Slide 6: Nonluminous infra-red heat lamp (black body radiator).

Slide 7.: Paraffin bath with thermostatic control.

Slide 8: Home made paraffin bath. Note that the thermometer is not needed and the temperature of the bath will not get too high if a piece of unmelted paraffin is kept in the bath. The patient plunges his hand in, quickly takes it out, and after a few seconds plunges it in again. This is repeated several times.

Slide 9: Home made paraffin bath.

Slide 10: Method of application of hot paraffin to back.

Thermotherapy: General Heat

Slide 11: General application of heat with the home made baker.

Slide 12: Electric light cabinet bath.

Slides 13, 14, 15, 16: Another method for the application of heat is by means of the full wet pack.

Slide 17: Electric blanket for provision of mild systemic heat.

Slide 18: A fever cabinet which employs hot circulating humid air for production of artificial fever.

Slide 19: Cabinet for production of artificial fever showing also an indicating thermometer.

Slide 20: Fever cabinet which employs short wave diathermy during induction period.

Slide 21: The emergency equipment for employment during administration of artificial fever.

Slide 22: Chart showing type of artificial fever curve recommended for treatment of resistant gonorrhea. (Temperature approximately 105.8 to 106.2 F. for eight hours.)

Slide 23: Table showing typical leukocytic response to fever therapy (febrile hemogram).

Thermotherapy: Cold

Slide 24: Trough in which ice is placed for refrigeration therapy of lower extremity.

Slide 25: Showing same trough as No. 24 filled with ice.

Slide 26: Improvised refrigeration device to be used with coil through which refrigerant is circulated.

Slide 27: Coil for local application of cold to an extremity.

Slide 28: Elaborate refrigeration device for application of cold to an extremity.

Electrotherapy: Galvanic

Slide 29: A portable instrument for provision of galvanic and faradic currents, useful in testing for reaction of degeneration.

Slide 30: A button electrode with finger interrupter as employed in testing for reaction of degeneration.

Slide 31: Motor point chart.

Slide 32: Motor point chart.

Slide 33: Arrangement for application of histamine by ion transfer.

Faradic

Slide 34: Diagrammatic representation of an induction coil for the production of the so-called "faradic" or "tetanizing" current.

Slide 35: Bristow's technic for producing graduated muscle contractions by means of the faradic current in muscles with a normal nerve supply.

Electrotherapy: Short Wave Diathermy

Slide 36: Another method of application of heat which is by means of short wave diathermy. This illustrates the cuff method of application for the high frequency electric field.

Slide 37: Cable method of application of the inductive field short wave diathermy. The cable is wrapped around the knee in this instance.

Slide 38: Another method of giving inductive heating by short wave diathermy. This is usually called a "pancake" coil.

Slide 39: Short wave diathermy method of application of a pancake coil to the shoulder.

Slide 40: Pancake coil beneath pillow.

Slide 41: Shows the air-spaced method of applying short wave diathermy.

Slide 42: Method of application of pad electrodes with short wave diathermy.

Slide 43: Diagrammatic illustration of panel board of electrosurgical unit.

Electrotherapy: Light Therapy

Slide 44: Technic of general ultraviolet bodily irradiation. Dosage factors are time and distance.

Slide 45: Patients receiving solar irradiation on a sun porch.

Slide 46: Carbon arc solarium.

Slide 47: Water-cooled ultraviolet lamp.

Slide 48: Quartz applicators for use with ultraviolet lamp.

Hydrotherapy

Slide 49: Contrast bath consisting of two buckets of hot and cold water. The extremity is immersed alternately in the hot and cold water.

Slide 50: Arm whirlpool bath.

Slide 51: A portable whirlpool bath.

Slide 52: A needle shower.

Slide 53: The Scotch Douche and needle shower.

Slide 54: Demonstration of Archimedes principle. A body submerged in water will lose as much weight as the weight of the displaced water. The principle on which under-the-water exercises are based.

Slide 55: Patient receiving treatment in a Hubbard tank.

Slide 56: Hubbard tank with overhead carrier.

Slide 57: Method of placing patient in Hubbard tank with the aid of a sling suspended from an overhead pulley.

Slide 58: Method of placing patient in therapeutic pool by means of a stretcher operated from an overhead hoist.

Kinesitherapy: Massage

Slide 59: There are four methods of giving massage: stroking movements (superficial stroking and deep stroking massage); compression movements (kneading, friction, percussion movements (clapping, hacking, slapping, tapping, and heating); vibration and shaking. Position of the hand for stroking and kneeding massage. Heat, massage and exercise are the major procedures in physical therapy.

Slide 60: Massage, stroking or effleurage.

Slide 61: Massage, kneading or petrissage.

Kinesitherapy: Therapeutic Exercise

Slide 62: There are any number of specific exercises for specific diseases. Buerger-Allen exercises (position one).

Slide 63: Buerger - Allen Exercise (position two).

Slide 64: Buerger - Allen Exercise (position three).

Slide 65: Powdered board to overcome friction during therapeutic exercise.

Slide 66: Gravity exercises in fracture of upper end of humerus. Arms should hang loosely and swing like a pendulum, shoulders and trunk being held immobile. Starting with a small arc, range should gradually be increased.

Slide 67: Sling suspension being employed for exercise of arm.

Slide 68: Simple overhead pulley for passive exercise of an arm.

Slide 69: Method of providing passive exercise for upper extremity by means of sling suspension and overhead pulleys.

Slide 70: Sling suspension and roller shoe for mobilization of lower extremity.

Slide 71: Sling suspension device for exercise of a lower extremity.

Slide 72: Use of Bradford frame and overhead pulleys to provide mobilization of lower extremities.

Occupational Therapy

Slide 73: Sanding to improve motion of upper extremity.

Slide 74: Sheet metal work. Patient employing metal shears for exercise of hand and wrist.

Slide 75: Employment of wall loom and sling for exercise of a shoulder.

Slide 76: Bicycle jig-saw.

Mechanical Appliances

Slide 77: Instruments for measuring motion in joints: a, for shoulder; b, for abduction and adduction of wrist; c, Wood protractor, and d, metal protractor.

Slide 78: The Council on Physical Therapy has developed a number of devices for exercise that

can be made by the local hospital carpenter. This slide presents specifications for construction of a shoulder wheel and abduction ladder which can be employed to improve shoulder extension, flexion, and abduction. Specifications for the construction of such apparatus can be obtained by writing to the Secretary of the Council on Physical Therapy of the American Medical Association.

Slide 79: A Kanavel table in use.

Slide 80: Stationary bicycle.

Slide 81: Stall bars.

Slide 82: A simple bed bicycle to be employed especially post-operatively for the prevention of embolism.

Slide 83: Sayre's head sling being employed for cervical traction.

Slide 84: Simple walker constructed from galvanized iron pipe. Specifications can be obtained by writing to Secretary of the Council on Physical Therapy, American Medical Association.

Slide 85: A foot inversion tread.

Slide 86: Mechanical tilting bed for treatment of circulatory diseases.

Slide 87: Suction pressure device for treatment of peripheral vascular disease.

Slide 88: Splint for radial nerve palsy (Buerki) and splint for prevention of toe drop.

Slide 89: Device for prevention of foot drop.

Slide 90: Also device for prevention of foot drop.

Slide 91: Device for providing passive flexion of the fingers.

Orthopedic Conditions

Slide 92: Cast for wrist, showing method of cutting cast in order to provide free motion for fingers and thumb.

Slide 93: Elastic traction device for production of fixed extension of wrist.

Slide 94: Proper method of application of airplane splint.

Slide 95: Cast for arm and forearm permitting shoulder exercises.

Slide 96: Plaster cast for functional decompensation of the back. Spinal and abdominal exercises are practiced regularly.

Slide 97: A corrective shoe showing a metatarsal bar and lift on the inner side of the sole.

Slide 98: Inter-relation of physical therapy with other methods of medicine in the treatment of arthritis. This reproduction is from the A. M. A.'s "Exhibit on Rheumatism" prepared by the American Committee for the Control of Rheumatism, Cooperating with the Committee on Scientific Exhibit of the American Medical Association.

CORRESPONDENCE

Re Medy — No Remedy

To the Editor: I have read with interest several recent communications in this department pertaining to a substitution for the designation of Physical Therapy. The last contributor who submitted the suffix "medy" would be ingenious if he were not so amusing, in fact so much so that I ask you to leave the pun I used as a heading "as is."

Being merely an honorary member of the Congress and therefore an outsider, I see some of your problems perhaps better than an insider, and I am inclined to blame the organizers of the original College (renamed Congress) for having missed the bus when they adopted the name "Physical Therapy." Supposing we use for purpose of illustration three of the numerous medical specialties, urology, neurology and cardiology. Do you think their followers would remain content to be called urologic, neurologic or cardiologic therapists? Decidedly not. They would yell at the top of their voices that they are not mere therapists, that they first of all diagnose their cases, determine the underlying pathologic factors and then select and carry out the indicated therapeutic measures. What, to use another comparison, would you think of a surgeon who described himself as a mere "operator?" Yet that is precisely what the term Physical Therapist implies, as a result of which many members of the profession this very day look on physical therapists as glorified physical therapy technicians. That is also why in many hospitals patients are referred to the physical therapy department by other clinicians much as a medical man refers a patient to whom he gives to a pharmacist to fill a prescription.

I have not the time nor the means to look up the back numbers of the ARCHIVES, but I have a distinct recollection of an editorial on this problem. In that editorial Dr. Kobak suggested the terms "physiatry" and "physiatrist," and in my opinion they designate etymologically and factually what scientific physical therapists should be recognized as—physicians specializing in physical medicine.

GUSTAVUS M. BLECH.

BOOK REVIEWS

THE 1943 YEAR BOOK OF PHYSICAL THERAPY. Edited by *Richard Kovács,* M.D., Professor of Physical Therapy, New York Polyclinic Medical School and Hospital; Attending Physical Therapist, Manhattan State, Columbus and West Side Hospitals; Visiting Physical Therapist, Department of Correction, Hospitals of New York City and Harlem Valley State Hospital, Wingdale; Consulting Physical Therapist, New York Infirmary for Women and Children, Mary Immaculate Hospital, Jamaica, N. Y., St. Charles Hospital, Port Jefferson, L. I., and Hackensack Hospital, Hackensack, N. J. Cloth. Pp. 409 with 75 illustrations. Price, $3.00. Chicago: The Year Book Publishers, 1943.

This volume is one of the 12 comprising the Practical Medicine Series of Year Books founded in 1900. No one is better fitted than Kovács to edit the year book on physical therapy. This book, coming at the close of the second year of American participation in the war, is timely because more than ever before army and civilian hospitals are operating fully staffed departments of physical and occupational therapy.

The book is divided into two parts. Part one is on Physical Therapy Methods and includes abstracts of the year's papers on thermotherapy; electrotherapy; light therapy; hydrotherapy and spa therapy; mechanotherapy, occupational therapy and institutional work.

Part two on Applied Physical Therapy considers rehabilitation; cardiovascular conditions; peripheral vascular disease; respiratory conditions; arthritis and rheumatoid conditions; traumatic, surgical and orthopedic conditions; poliomyelitis, spastic paralysis, peripheral nerve injuries; neurologic and mental conditions, gynecologic, pediatric, genitourinary conditions; syphilis and gonorrhea, proctologic, dermatologic and ophthalmologic conditions.

Attention is directed to the fact that hypothermy has found a widening scope of usefulness in military and industrial surgery and in peripheral vascular disease; the rationale and technic of refrigeration anesthesia for amputations have been fully developed. The now common wartime injuries due to cold have been subjected to much study and a variety of clinical procedures has been developed. Fever therapy has been routinely employed in combination with chemotherapy in cases resistant to the latter method alone. Modern methods of exercise, systems for increasing bodily fitness and for measuring muscle development which have commanded attention on both sides of the Atlantic are considered in this volume. Rehabilitation and the correlation of occupational therapy with physical therapy are stressed. The mode of action of air sterilization by ultraviolet rays has been abstracted. Physical treatment of spastic paralysis and nerve injuries are given extensive study. It is shown that electric shock therapy of the psychoses was widely used clinically.

Every recent method of physical therapy is presented in this excellent yearbook. It can be highly recommended to physicians interested in physical medicine in the armed forces or in civilian life, to physical and occupational therapy technicians.

FRACTURES AND DISLOCATIONS FOR PRACTITIONERS. By *Edwin O. Geckeler,* M.D., Fellow of the American College of Surgeons, Fellow of the American Academy of Orthopaedic Surgeons, Diplomate of the American Board of Orthopaedic Surgery. Third edition. Cloth. Price, $4.50. Pp. 361, with 320 illustrations. Baltimore: William Wood & Company, 1943.

The purpose of this book is to condense the subject of fractures and dislocations without the omission of important details and is intended to fulfill the need for a complete, yet simplified, guide to the management of bone and joint injuries. In the preface to the third edition the author states that the increasing importance of traumatic surgery, and new developments in the treatment of fractures, call for another edition of the book. As many of our surgeons have gone to war, the physicians who remain must assume additional duties which include minor surgery and the care of fractures; likewise, the increase in accidents due to expansion of industry places an additional burden on them.

The care of fractures is one of the most important duties of the general practitioner, yet many of them do not understand the fundamentals of the subject. Fractures are the source of much anxiety to the medical profession; the public expects a perfect result and unless the patient recovers completely there is a great deal of dissatisfaction. The delay on account of lack of understanding, faulty judgment, and neglect to apply common sense principles explain most of the unfortunate results. This book is written for the general practitioner; it stresses the application of common sense principles, from correct initial judgment to modern methods of treatment.

New procedures have been simplified, and the sections on emergency treatment and fracture wounds, which are vitally important at the present time, have been prepared from the current war literature. Considerable attention has been given to chemotherapy, recent developments of which have practically revolutionized the treatment of traumatic wounds.

The book is divided into two parts, fractures and dislocations. In the various chapters are described the fundamentals concerning the examination, diagnosis, records, medicolegal aspects, complications of fractures and dislocations and the organization of fracture services. Regional fractures are discussed,

followed by several chapters on general considerations and specific description of the reduction and treatment of dislocations. This edition can be recommended with as much confidence as its predecessors, especially to the general practitioner.

PRINCIPLES AND PRACTICE OF REHABILITATION. By *John Eisele Davis*, M.A., Sc.D., Veterans Administration Facility, Perry Point, Maryland, Cloth. Pp. 211. Price, $3.00. New York: A. S. Barnes & Company, Inc., 1943.

This volume deals with rehabilitation in the field of mental illness. In this field the problem of rehabilitation is most complex and involves large numbers. There are approximately as many people receiving treatment in mental hospitals or care in institutions for mental defectives as there are in all the general hospitals in this country. Half a million men, women and children will enter mental hospitals in the next five years and as many more are already there. The cost to the public is a quarter of a million dollars a day. Modernized methods are bringing increased numbers from the ranks of the mentally ill into focus of rehabilitation.

The present war will add further to this number who will require the services of rehabilitation. There is no way of estimating the percentage of mental and physical handicaps which will result to the millions in actual combat. War neuroses present a tremendous military problem for modern warfare. The ravages of war do not produce all the mental and physical handicaps during the active phase. After the period of excitement during demobilization, the greater number of neuroses develop. These conditions are long drawn out, and tend to increase for at least twenty-five years after the cessation of hostilities. This increase in the precipitating factor and the increasing understanding of society at large of its responsibility to restore the injured and handicapped to a social and economic status through rehabilitative methods are factors which will add enormously to the growing field of modern rehabilitation.

It should be read by physicians engaged in rehabilitation in civil life and in the army forces it should be read by all physical and occupational therapy technicians.

THE HOSPITAL IN MODERN SOCIETY. Edited by *Arthur C. Bachmeyer*, M.D., Director, University of Chicago Clinics; Director, Hospital Administration Course, University of Chicago; and *Gerhard Hartman*, Ph.D., Director, Newton Hospital, Newton Lower Falls, Mass. Cloth. Pp. 768. Price, $5.00. New York: The Commonwealth Fund, 1943.

This is a collection of readings selected by the editors from the literature in the hospital field, and in the allied fields of medicine, public health management and organization, law, sociology and psychology. The editors state that the material was taken exclusively from the periodical literature and from transactions and committee reports, because they felt that the few books in the field would be in the libraries of persons professionally interested.

This book is intended for students of hospital administration who are about to enter the field professionally and who wish to correlate their observations with an organized approach to the more important problems in the field. A student it is felt, would get something more from this book if after each author's name of an abstract the editors would put his official connections, for instance, "The Hospital and the Radiologist by Robin C. Buerki" would be more authoritative to the student who might not know Dr. Buerki, if the editors had inserted after his name "Dean of the Postgraduate Medical School of the University of Penna., and Director of the University and Postgraduate Hospitals."

The selection of the material is excellent and each article gives a judicious analysis of its problem and stimulates thought. Among the authors are men and women who may be numbered among the deans of hospital administration. Ninety-eight authors are represented by the 145 articles. Physical Therapy physicians will be interested to note that an article is given on "Physical Therapy Department in Small, Medium and Large General Hospitals."

A thing that adds to the value of this book is the lists of "References for Further Reading" after each subject. These serve as aids to students of hospital administration for further exploration of a particular subject.

This volume is recommended to (1) hospital administrators; (2) hospital department heads and (3) to students in hospital administration.

THE FOOT. By *Norman C. Lake*, M.D., M.S., D.Sc. (Lond.), F.R.C.S. (Eng.). Senior Surgeon and Lecturer on Surgery, Charing Cross Hospital; Surgeon, Bolinbroke Hospital; Consulting Surgeon, Emergency Medical Service; Director of Studies, London Foot Hospital. Third Edition. Cloth. Pp. 432, with 136 illustrations. Price, $5.00. Baltimore: The Williams & Wilkins Company, 1943.

In this new edition fresh material has been added to bring it up to date for wartime, such as gun-shot wounds, trench foot, immersion foot, and foot fatigue in industry and the armed services. There are also changes in the etiology and mechanism of gait. The author has added at the end of the book "Reference Books and Papers" which includes those written in the United States as well as in Great Britain.

A book on diseases and injuries of the foot should be read by all physicians both in the armed forces and in civilian life, because there is much incapacity in addition to social discomfort caused by foot disorders. Frequently the responsible lesion is a comparatively trivial one and the sufferers find that their complaint receives but scant attention when they present themselves for advice. Too often an overcrowded medical curriculum prevents a medical student from receiving instruction in such apparently minor matters, although he discovers later that they constitute a considerable portion of his practice.

This monograph deals with those conditions which are likely to come into the care of the general practitioner and most of the major orthopedic conditions have been deliberately omitted. It is therefore recommended as a good book for the general practitioner to have in his library.

PUBLIC HEALTH NURSING. *Pearl McIver,* Principal Nursing Consultant, United States Public Health Service, Division of States Relations. Paper, Supplement No. 133 to the Public Health Reports. Revised. Price, 10 cents. Washington, D. C.: U. S. Government Printing Office, 1943.

Since there is an urgent need for physical therapy technicians to qualify in public health work those technicians who are nurses will find this brief résumé an informative guide. Many of the services are outlined. Qualifications are set forth, and the administration and future of public health nursing and services are discussed.

BIOCHEMISTRY FOR MEDICAL STUDENTS. By *William Veale Thorpe,* M.A., Ph.D. Third edition. Fabrikoid. Price, $4.50. Pp. 476, with 39 illustrations. Baltimore: William Wood & Company, 1943.

The aim of this book is to present an account of the biochemical processes known to occur in the healthy human body. In view of their importance in medical diagnosis, prominence has been given to blood, urine and faeces. Special attention has been paid to the principles of nutrition and the composition of foodstuffs. The third edition retains the valuable features of the earlier editions. A considerable amount of new material has been added including a short chapter on respiration. In setting forth the plan of this book the author states that all living processes are carried out in aqueous solution, so that as a preliminary the physicochemical principles involved are reviewed, chapters 2 to 4. Chapters 5 through 9 present study of the commoner substances found in our tissues and food. Other substances of physiologic importance are described in chapters 12, 13, 20, 25 and 26. In chapters 10 and 11 there is discussed the subject that living tissues are provided with special catalysts which facilitate and control various chemical reactions. The distribution of the different substances in our tissues is given in chapters 13 and 21.

In part 2, details of the better known metabolic processes are discussed, showing how food is digested and absorbed into the body and changes which substances undergo in the tissues; intermediary metabolism, chapters 14 through 24. Other chapters describe how the body uses atmospheric oxygen and excretes carbon dioxide, hormones and vitamins. Part 3 covers the study of the body as a whole, its energy needs, the form and conditions under which food should be taken, the composition of food and the waste products eliminated from the body. The book is intended to be complementary to textbooks of physiology and contains sufficient material in condensed form to enable one to understand physiologic and biochemical processes.

AN ATLAS OF ANATOMY IN TWO VOLUMES. By *J. C. Boileau Grant,* M.D., M.B., Ch.B., Professor of Anatomy in the University of Toronto, Toronto. Volume II. Vertebrae, and Vertebral Column, Thorax, Head and Neck. Cloth. Price, $5.00. Pp. 390, with 232 illustrations. Baltimore: William Wood & Company, 1943.

The collection of illustrations depicts the structures of the human body, region by region, in much the same order as the student displays them by dissection. Volume II follows the same method of presentation as does volume I. In general it should be pointed out that these two volumes can serve as an excellent supplement to the laboratory work of the student and should save the student considerable time. This volume like volume I is highly recommended.

WAR ENDOCRINOLOGY. By *James H. Hutton,* M.D. Cloth. Pp. 363. Chicago: Wayside Press, 1943.

The average general practitioner of the older generation looks bewildered at the mass of research material developed in the field of endocrinology in recent years and has a somewhat nebulous conception of endocrine disturbances in general. He may recognize the occasional advanced case but has difficulty in diagnosing and treating many of the milder or incipient cases. The author has been active in the field of endocrinology for many years and offers his volume under the somewhat far-fetched title as a contribution to the maintenance of national health at a high level during war time. His aim is to provide a practical and concise manual of information to the older physicians in private practice who cannot familiarize themselves with the voluminous literature. Twelve successive chapters present The Pituitary, The Thyroid, The Adrenals, Female Endocrinology, Male Endocrinology, The Gastro-Intestinal Tract, Essential Hypertension and Diabetes, The Parathyroids, The Thymus and the Pineal, Obesity, Methods of Study, Endocrine Preparations. There are no illustrations, no lengthy statistics and there is no bibliography. However, there are quite a few illustrative case histories, the material is presented in a conservative manner and a well readable style. The author has fully carried out his professed purpose and his book should serve as a welcome guide to physicians through the maze of modern endocrinology.

PHYSICAL THERAPY ABSTRACTS

Refrigeration Anesthesia in Amputations. Harry E. Mock, and Harry E. Mock, Jr.

J. A. M. A. **123**:13 (Sept. 4) 1943.

This article comprises a critical analysis of the literature on refrigeration anesthesia for amputations based on personal experience. To the 101 cases reported by Allen, Crossman and others, the authors add observations on 17 cases of their own, eight amputations for peripheral vascular disease and nine amputations for trauma.

By refrigeration is meant the chilling of tissues, not freezing. Freezing damages tissues as in frostbite; refrigeration does not. Water freezes at 0 C. (32 F.). Blood and tissues freeze at a slightly lower level. Refrigerating a limb with cracked ice or ice water lowers the temperature to somewhere between 0.5 and 5 above freezing. With a mechanical device the exact degree of refrigeration may be controlled. Hence there is a definite margin of safety. In no instance have we seen the tissues damaged.

In an effort to determine the exact temperatures deep in the tissues in various locations of the refrigerated extremity, with and without the use of a tourniquet, internal temperature measurements have been made in two most recent amputation cases. These readings were made with an L. & N. potentiometer, hypodermic type No. 1, thermocouple.

In the authors' experience a slight delay in healing has been the only disadvantage encountered when refrigeration anesthesia is used for amputations. Its advantages are many to overcome this objection. Postoperatively there is no pain; none of their patients have required an analgesic. There is no shock or other reaction to what is commonly considered a rather shocking operation; the blood pressure, pulse and respiration show no change during or after the operation. It permits surgery to be performed in what formerly were hopeless cases because of debility or septicemia. It lowers the incidence of stump infections in those limbs requiring amputation because of infection. It is of especially great value for diabetic patients when the absence of any postoperative reaction allows the patient to carry on without missing a meal. The diet and insulin dosage do not have to be juggled.

In severe crushing injuries of an extremity or other injuries with complications necessitating amputation, refrigeration will inhibit infection and limit the absorption of histotoxins, thus helping to overcome shock, and will give time for infusions of plasma, blood or fluids and other necessary preparatory measures. With refrigeration the amputation may be delayed until life threatening associated injuries or complications have been conquered. Finally, under refrigeration anesthesia the extremity may be amputated at the selected site without further shocking the patient.

Rate of Regeneration of Peripheral Nerves in Man. H. J. Seddon; P. B. Medawar, and H. Smith.

J. Physiol. **102**:214 (Sept. 30) 1943.

The rate of regeneration of peripheral nerves in man has been so universally accepted at about 1 mm. a day that the basis for and accuracy of this estimate have long remained unchallenged. There is no doubt that the rate is something of this order as compared with figures such as 0-1 or 10 mm. a day. But the subject is a complex one. There may be different rates for fibers of different types, and it is probable, even likely, that the various methods of estimation at our disposal will give results that are not even comparable. Is it true that regeneration progresses at a uniform rate throughout the length of a nerve?

The paper describes obesrvations on the rates of recovery of sensory and motor functions in man after peripheral nerve lesions of different types.

After the interruption of a nerve, the muscles normally supplied by the part that lies peripheral to the lesion return to functioning in the anatomical order of their nerve supply. An estimate of the rate of motor recovery may be obtained by recording the times at which the muscles show their first signs of voluntary contraction, and by making use of specially collected anatomical data showing the distances at which they lie from the level of the interruption.

The rate of sensory recovery may be calculated by measuring the advance of pain and touch sense in a long zone of cutaneous insensibility.

The rate of advance of Tinel's sign, though of limited functional significance, has some prognostic value and provides an additional method for the study of sensory recovery.

The rate of recovery falls off progressively as the process moves towards completion. It can, however, be regarded as constant over the moderate ranges of time and distance over which the process was recorded in the great majority of the cases described. The following average estimates have been obtained for the rate of motor recovery. In the radial nerve: after suture 1.6 $\pm$ 0.2 mm. day; after axonotmesis 1.5 $\pm$ 0.1 mm. day. In all nerves studied: after suture 1.5 $\pm$ 0.2 mm. day; after axonotmesis 1.4 $\pm$ 0.1 mm. day. The average rate of advance of Tinel's sign after suture has been found to be 1.7 mm. day. Other estimates of sensory and motor recovery have been calculated from less complete data of our own and from the data of Stopford.

The principal variables that affect clinical estimates of the rate of recovery are, after suture: the interval between injury and repair of the

nerve, the state of the stumps at the suture line and the amount of damage inflicted by post-operative stretching in cases where extensive resection before suture had proved to be necessary; after axonotmesis, the extent of the damage to supporting structures within the trunk of the nerve.

When estimates have been corrected for the greater range of times and distances over which recovery in human cases can be recorded, the rates of recovery in man are not found to be greatly different from those estimated by more precise methods in the rabbit.

Studies of the rate of recovery in man are of value for the prognosis of nerve injuries and for judging the efficiency of the various types of operation used for their repair.

The Ultraviolet Absorption Spectra of Lignin Compounds. R. F. Patterson, and Harold Hibbert.

J. Am. Chem. Soc. **65**:1873 (Oct.) 1943.

The behavior of lignins as absorbers of energy in the ultraviolet region has been compared with that of known compounds in the same region and certain conclusions drawn from such comparison. These have been enumerated and may be briefly summarized as follows: The spectra of amorphous lignins can be explained satisfactorily on the assumption that they are derived from lignin precursors of the type exemplified by hydroxy derivatives of 1-(4-hydroxy-3-methoxyphenyl)1-propanone and 1-(4-hydroxy-3, 5-dimethoxyphenyl)-1-propanone. The absorption curves indicate that lignin is aromatic in nature. The evidence suggests that a carbonyl group of an ethylenic double bond is present in conjugation with the aromatic nucleus to some at present unknown extent.

Demonstration of Apparatus for Self-Activated Exercises in the Early Rehabilitation of the Sick and Injured. O. F. Guthrie Smith.

Proc. Roy. Soc. Med. **36**:610 (Sept.) 1943.

There is confusion of thought between reeducation of function, remedial exercises and rehabilitation.

In the early stages of the reeducation of function the patient may require help either in the form of manual assistance or simple aids such as suspension slings and pulleys. Such assistance calls for precise technic in order to localize the effort to the weak muscles and to upgrade the strength of exercise as function improves.

The Swedish system of remedial exercises is characterized by manual assistance or resistance in which the operator works on the patient rather than by teaching the patient to help himself; but this type of manual and individual treatment cannot be sustained for sufficiently long periods nor can it provide the tempo necessary to make a patient fit for strenuous occupations.

In rehabilitation the function of the technician is to teach the patient to carry out his own exercises, to understand the purpose of the work and to cooperate by stimulating mental as well as physical effort. To this end apparatus has been designed which is activated by the efforts of the patient or a group of patients working together, whilst suspension slings and springs are employed to localize the muscle work and to give a predetermined degree of assistance or resistance; effort and rest periods alternate in rhythm.

A Muscle Testing Board. Mary Eleanor Brown, and Robert L. Thompson.

Physiotherapy Rev. **23**:156 (July-August) 1943.

Physicians, physical theray technicians, nurses, teachers and students of muscle testing may welcome the suggestion of a muscle testing board which is stable and needs no additional hands to steady it.

The muscle testing board has the advantage of being firm even if placed on a mat or blanket because it has legs attached to it and therefore no special holding or manipulation of it is necessary.

The specifications for making the board are given.

The Irradiation of Abietic Acid With Ultraviolet Rays. Ronald F. Brown; G. Bryant Bacham, and Stanley J. Miller.

J. Am. Chem. Soc. **65**:626 (April) 1943.

Unfiltered ultraviolet light has been shown to have no effect on abietic acid when in solution in hexane or benzene. Abietic acid has been shown to be oxidized to dihydroxyabietic and tetrahydroxyabietic acids when an alcoholic solution was irradiated with unfiltered ultraviolet light. If the light was filtered through alcohol, the abietic acid remained unchanged. An improved procedure for the separation of abietic acid, dihydroxyabietic acid and tetrahydroxyabietic acid by solvent extraction has been devised.

The Mechanism of Valgus Foot Strain. E. J. Crisp.

Proc. Roy. Soc. Med. **36**:607 (Sept.) 1943.

Until the mechanism producing valgus foot strain is better understood treatment will remain unsatisfactory, and the invariable prescription of foot class exercises and faradic foot baths lead to many disappointments.

In its early stages valgus strain is easy to remedy, but when it becomes complicated by failure of the normal toe function, restoration becomes difficult.

The primary cause of valgus strain is fatigue of the tibialis anticus. This muscle supports the long arch during weight-bearing and progression, and holds the foot dorsiflexed when it is off the ground as in stepping forward. In its former action it is assisted by the tibialis posticus and in the latter by the extensor digitorum longus.

Among the contributory factors responsible for

its breakdown are faulty posture, adolescence, overweight and in the Army, heavy boots and long marches. Failure of the muscle leads to flattening of the long arch during activity, though when off the ground the foot is still supported and dorsiflexed by the extensor digitorum longus.

Pain results from stretching of the plantar fascia. Rest in bed until this is relieved, followed by correction of posture, reeducation in walking and retraining of the tibialis anticus will correct the condition.

The Fluorescein Test in the Management of Tubed (Pedicle) Flaps. James A. Dingwall, 3rd, and Jere W. Lord, Jr.

Bull. Johns Hopkins Hosp. **73**:131 (Aug.) 1943.

By means of the fluorescein test the earliest possible time for transfer of a tubed flap can be determined and the transfer of a pedicle carried out with assurance that the circulation is adequate. The test is simple to perform and apparently safe in the dosage necessary for accurate determination of color changes. It is suggested that smaller doses should be used in handling children.

Some Observations on the Adaptation of the Double Beam Cathode-Ray Oscilloscope to Biology and Medicine. G. E. Donovan.

Proc. Roy. Soc. Med. **36**:606 (Sept.) 1943.

The cathode-ray tube is used as a recording device in the fields of medicine and biology. A practical application of the multiple recording of nerve action potentials is electroencephalography. Muscle action potentials are assuming a greater diagnostic significance in the investigation of certain neuromuscular affections. Multiple recording helps in the accurate localization of the lesion.

The author has shown elsewhere some of the uses of the double beam cathode-ray oscillograph in cardiology.

The rate of propagation of the pulse is of value in assessing the elasticity of the arteries. The hot wire sphygmograph has been used for this purpose; simultaneous records being taken of the central pulse and also the peripheral. In latter years a photo-electric plethysmograph has proved its usefulness in recording the flow of blood through a peripheral part. The variations of the flow of blood cause changes in intensity of a beam of light which is being transmitted through the part. These changes in light are converted into an electrical equivalent by a photo-electric cell which can be amplified and demonstrated in oscillographic form. If an electrocardiogram is recorded as one trace of the double beam cathode-ray oscillograph and the amplified output of the electrical variations of the photo-electric cell is recorded by the second beam, the rate of propagation of the pulse can be estimated.

The piezo-electric manometer has been used by American workers for studying intracardiac pressure and at the same time the simultaneously recorded intravardiac electrograms. The double beam cathode-ray oscillograph lends itself readily for the recording of these phenomena.

Paralytic Scoliosis. Aladar Farkas.

J. Bone & Joint Surg. **25**:581 (July) 1943.

During the epidemic of infantile paralysis in the State of Iowa in 1940, 264 patients with this disease were admitted to the Orthopedic Department of the State University of Iowa. In more than 100 patients, as soon as the condition of the patient permitted, roentgenograms of the spine were taken, regardless of the muscle involvement and the severity of the disease. The examination and evalution of this material and its comparison with more than 100 patients in previous epidemics form the basis of the paper.

The author observes that in most patients from four to five years are necessary for the complete development of paralytic scoliosis. Only after that period can a scoliotic curve be regarded as mechanically final; this also marks the end of the early stage.

A few weeks or months after the onset of infantile paralysis, the spine discloses changes representing a pathologic entity, the paralytic spine. This process can be subdivided into several stages characterized by (1) morphologic and (2) functional signs.

At the onset enlargement of the intervertebral spaces is observed, followed later on by dullness and cloudiness; the border lines between discs and vertebrae are effaced; and the spaces themselves become uneven and appear markedly narrowed.

Functionally the paralytic spine is characterized by a high degree of flexibility and compressibility. Because of the increased mobility, univertebral or segmental rotations appear first without any lateral deviation.

The paralytic spine is the pathologic condition preceding paralytic scoliosis. The scoliotic curve requires usually from four to five years before reaching its final form. Prior to this, the side of the convexity and the direction of the rotation may change several times. The rotation and compression of the spine are the chief factors in preparing the way for paralytic scoliosis.

The rotation is brought about by faulty mechanics of (a) the pelvis; (b) the thorax and shoulder girdle (thoracic rotation); and (c) the respiration.

In pelvic rotation all spinous processes point to the same side of the body. In thoracic rotation, the thoracic and the lumbar spinous processes point in opposite directions.

The cause of rotation is the pathologic imbalance between the two sides of the body in carrying out rotary motions of different degrees during the performance of the daily routine, especially during locomotion. The physiologic imbalance, present in every human being, takes advantage of the decreased resistance of the rotary system of the spine and causes the predominance of the right thoracic, left lumbar curves.

Paralytic scoliosis is brought about by the imbalance between the two sides of the body exerted on the paralytic spine. Paralytic scoliosis can be differentiated from a scoliosis of any other etiology by the uniform density of the spine in the roentgenogram, by the excessive and early rotation of the vertebrae, and by the temporary concave rotation.

Curves resulting from pelvic rotation, except for the sitting curves, have a far better prognosis than the thoracic curves, especially if the latter are associated with respiratory disturbances.

Objective Methods to Determine the Speed of Blood Flow and Their Results (Fluorescein and Acetylene). Kurt Lange, and Linn J. Boyd.

Am. J. Med. Sci. **206**:448 (Oct.) 1943.

The appearance of fluorescein in the lips under a special ultraviolet light can be used to determine the circulation time, but certain conditions must be observed in order to obtain reliable results. In 212 normal adults, the values for the fluorescein circulation time ranged between 15 and 20 seconds, the majority between 15 to 17.5 seconds. The average of the circulation time is longer in older patients. Work accelerates the circulation time considerably and may make it 2½ times faster than normal. Fever shortens circulation time. The fluorescein method can be used to determine the velocity to different points of the body. The average time to conjunctiva, lips, rectum, foot, is 10, 15, 18 and 23 seconds, respectively. The conjunctiva is not an appropriate place to test the circulation time. In congestive right heart failure 92 per cent of the cases show a prolonged circulation time, while compensated cases of heart disease have normal circulation times. Pure bronchial asthma, having a normal or slightly shortened circulation time, can be differentiated by the fluorescein circulation time from cardiac asthma, which has a prolonged circulation time. Hyperthyroidism is associated with shortened circulation time values, which seem to provide an earlier indication of the clinical situation than the basal metabolic rate. Patients with hypothyroidism have prolonged circulation times. Anemia considerably shortens the circulation time when the red blood cell count goes below 3,500,000. Inhalations of acetylene can be used to determine the time which elapses until all blood in rapid circulation has passed the lungs at least once. Work shortens the slowest circulation time as much as 2½ times the normal. The comparative values found with the fluorescein method for the fastest and the acetylene method for the slowest circulation time show the same relation.

A Statistical Study of Minor Industrial Burns. Roy D. McClure, and Conrad R. Lam.

J. A. M. A. **112**:909 (July 31) 1943.

Much has been written on the subject of burns during the past decade and a half, and a large amount of laboratory and clinical investigation has been carried out. Nevertheless at the present time there is no method of local treatment which is accepted as standard by those who take care of these injuries.

It has been suspected that there has been considerably less standardization of the treatment of minor burns, such as are seen frequently in industrial plants.

A study was made which brought out the striking fact that no less than eighty-four different substances were used in the treatment of the burns included in this experiment. Many of these remedies were proprietary preparations, some of which were adequately described on the labels or advertising literature; others were described ambiguously. There were several different brands of certain more or less standard substances, such as tannic acid jelly and vitamin ointments. Each brand was tabulated separately, which helped to swell the list of different materials to the high figure just mentioned.

In the treatment of such small injuries our aim should be to (1) prevent disability (get the man back on the job in a matter of minutes), (2) relieve pain, (3) prevent or minimize infection and (4) favor early healing, with a minimum of dressings. These objectives are different in many ways from those we have in mind when we are treating the serious burn in the hospital. In this instance the problem is to save the patient's life, which involves considerations of shock due to plasma loss, serious infection, scarring and contractures.

An unreasonable number of different preparations are being used for the treatment of minor burns in industrial plants. Regardless of what is put on the average minor industrial burn, it is apt to be healed in less than a week.

The authors recommend the following treatment for minor industrial burns: wash the area with white soap and water; do not break blisters or otherwise "debride" the wound; cover with fine mesh gauze impregnated with petrolatum or 5 per cent boric acid ointment; apply a firm dressing over this, bulky enough to keep dirt away from the injury but not too large to keep the man off his job.

Management of the Venereal Diseases in the Army. Thomas B. Turner, and Thomas H. Sternberg.

J. A. M. A. **124**:137 (Jan. 15) 1944.

During recent months the rate of venereal infection in the Army has been below preceding peacetime levels and less than half that recorded during the first world war. Days lost per thousand men annually have dropped from 1,278 in 1940 to a level of approximately 400 at the present time. Nevertheless venereal disease patients accounted for approximately 2,824,000 hospital bed-days during the twenty month period from January, 1942 to September, 1943.

From recent Army experience in the management of venereal disease, several conclusions may be drawn. First, while the treatment of syphilis has been greatly improved, no method of therapy yet developed is entirely satisfactory. The prob-

lem is of sufficient importance to justify speedy exploration of all new leads.

Secondly, biologic false positive serologic tests occur often enough after vaccination procedures and following acute febrile illnesses to constitute a serious diagnostic problem. Under such circumstances follow-up observations are necessary in order to avoid treating individuals for syphilis in the absence of infection.

Thirdly, the treatment of gonorrhea has become largely one of internal medication. Urethral instillations, prostatic massage and the passage of sounds are contraindicated in the early phase of the disease for they frequently induce complications.

Fourthly, penicillin promises materially to change the management of gonorrhea. When supplies of this drug become readily available, sulfonamide resistant gonorrhea should no longer be a problem, and the necessity for long continued treatment or for the use of fever therapy in these cases will have been eliminated.

Finally, certain administrative practices and social attitudes, valid perhaps in an earlier day, are now hampering the full application of the remarkable scientific advances made in the treatment of these diseases. Medical treatment is not so effective that diseased persons can promptly be rendered noninfectious and returned to duty as functioning members of the group. Any measures, therefore, which tend to keep the infected person away from medical care are directly opposed to the best interests of the individual, the organization to which he belongs and the community. Stoppage of pay, prejudicial treatment from employers or superiors, restrictions on advancement, can only serve as powerful factors tempting the individual to conceal his infection and delay treatment until its effectiveness is reduced. The sooner discriminatory practices are abolished and syphilis and gonorrhea regarded as other infectious diseases, the sooner will it be possible to realize the full benefits of recent scientific progress.

Electromyography in Clinical Medicine. Graham Weddell.

Proc. Roy. Soc. Med. **36**:514 (Aug.) 1943.

In 1929 Adrian and Bronk showed that the action potentials from voluntary muscle can be recorded by means of a concentric needle electrode. Denny-Brown and Pennybacker (1938) used a similar method in a study of fasciculation and fibrillation of voluntary muscle. The authors employed this method to obtain information concerning the electrical activity of voluntary muscle in a number of pathologic processes particularly those involving peripheral nerves (Weddell, Feinstein and Pattle, 1943).

By a series of animal experiments, they established the nature of the electrical activity present in voluntary muscles after denervation by complete crushing of the nerve and during the course of nerve regeneration until functional recovery has taken place.

It has been found that electrical activity is always present when there is muscle tissue. Where this has been replaced by fibrous tissue, no electrical activity whatever can be obtained.

Electromyography was found to be of aid in diagnosis and prognosis in the following conditions: In cases of peripheral nerve injury fibrillation action potentials in the absence of motor unit action potentials are indicative of a complete lower motor neurone denervation of the muscle. This is of particular value in those cases where the muscles fail to respond to percutaneous galvanic stimuli. In conditions of muscle weakness and wasting when a partial nerve interruption has occurred, e. g., cervical rib, a mixture of fibrillation and motor unit action potentials is found.

Following regeneration of a peripheral nerve, a mixture of fibrillation and highly polyphasic motor unit action potentials appears before signs of recovery can be determined clinically or before the electrical reactions alter.

The presence more than three weeks after injury of an outburst of motor unit action potentials on insertion of the electrode, and the absence of fibrillation action potentials, indicate that no axons have been severed, e. g., in certain cases of "Bell's palsy." In such cases prognosis is usually good. If fibrillation appears after three weeks, recovery is by regeneration with the development of associated movements. When no electrical activity whatever can be obtained from a muscle, it may be concluded that severe morphological changes have occurred, e. g., fibrosis. This sometimes occurs in peripheral nerve lesions where treatment of the muscle has been inadequate.

In certain cases where denervated muscles are in poor condition and fibrillation action potentials are difficult to obtain, even after warming, prostigmine is valuable in helping to elicit them.

A case of radial nerve palsy was shown and the fibrillation action potentials from the denervated muscles compared with the electrical activity from the normal side.

The Eccentricity of Standing and Its Cause. F. A. Hellebrandt; Bette G. Nelson and Eleanor M. Larsen.

Am. J. Physiol. **140**:210 (Nov.) 1943.

The early investigators in the field of animal mechanics assumed that in the well-formed individual the vertical projection of the center of gravity of the body as a whole must lie in the midsagittal plane (Schafer, 1900). Thus the human body in the normal Stellung of Braune and Fischer (1890) was represented as passively poised over the ankle joint in a strictly symmetrical posture. Although it is well recognized that gravity is the chief deforming force affecting the vertical alignment of man, the magnitude and disposition of the gravitational rotatory stresses have been little studied, and direct experimental confirmation of the symmetry of standing is difficult to find. We have observed that the ver-

tical projection of the center of gravity tends to fall slightly to the left and behind the geometric center of the total supporting base (Hellebrandt; and Braun, 1939; Hellebrandt and Fries, 1942). This posterosinistral stance eccentricity occurs with sufficient frequency to suggest that the deviation may have biological significance. The authors found it to be characteristic of about 80 per cent of the subjects thus far studied.

A series of experiments designed to yield quantitative estimates of right and left-sided differences in size, strength and limb preference were performed on a small group of young adult women in an effort to elucidate the mechanism of the slight posterosinistral eccentricity of the vertical projection of the center of gravity of the body as a whole which characterizes the upright stance of 80 per cent of normal subjects. The evidence substantiates the following conclusions:

1. Morphologic and functional asymmetries occur in limb preference, volume and strength.
2. Although most of the observed asymmetries are too small to have statistical significance they constantly favor the right side.
3. It is suggested that in the aggregate these small dextral asymmetries in functional capacity associated with like differences in strength and size have the effect of a slightly eccentric counterweight on the incessantly shifting rotatory moments acting on the joints of the weight-bearing skeletal parts. The autonomous equilibrating muscular contractions called forth overcompensate for the force of this eccentric weight and the anteriorly unbalanced position of the leg by an amount great enough to result in a slight eccentricity in the location of the mean vertical projection of the center of weight in a position contralateral to the sum of the unequal stresses.

Desert Sores. B. R. Sandiford.

Correspondence Brit. M. J. **4327**:590 (Nov. 6) 1943.

Sir: — The article by Dr. J. M. Henderson (May 29, p. 657) on the relation of sunlight to desert sores prompts me to put forward a possible aspect of this relation which has exercised my mind for some time.

The effect of the ultraviolet rays of the sun in decreasing the polymorphs, increasing the lymphocytes, and causing a "shift to the left" in the polymorph nuclei, has been shown by several workers. Shaw (1936) demonstrated the latter in Egypt and Kennedy (1935) in Iraq. Pellicciotta (1939) found neutrophilopenia and lymphocytosis with a shift to the left among white residents in Africa. Stammers (1933) says: "It is an established fact that ultraviolet irradiation leads to lymphocytosis." Does it not seem probable, then, that a neutrophilopenia due to solar irradiation, further increased in many instances by the common custom among the troops of sun bathing, may underlie the reduced resistance to infection of some trivial injury? It is interesting to note that Corkill (1939) in the Sudan recorded a relation between increased insolation and increased incidence of cerebrospinal meningitis. Although he postulated a vitamin deficiency as the connecting link, I feel that diminution in the polymorphs might have something to do with it.

As regards the treatment of desert sores, I should like to suggest the application of a wad of gauze soaked in sterile peptone broth as used in the bacteriology laboratory. Applied night and morning for a week or two it often works wonders in cleaning up dirty ulcers and promoting healing. Its efficacy makes me wonder whether the so-called "ultra-virus" preparations may not owe their action not so much to the products of bacterial growth as to the peptone broth which forms their basis. In some cases, after initial improvement an ulcer will again become "indolent," in spite of continued use of the broth. This accords with the experience of Rapport (1942), who, from his experience of treating desert sores, found that it was not advisable to persevere for longer than two weeks with any particular treatment, as the sores tended to acquire a tolerance to it. How the broth acts I do not know; possible peptone water, or meat extract, or even saline dressings would work equally well. I have not access to sufficient cases to determine the question.

Rise of Potassium Concentration in the Blood Stream Following Ischaemia of Muscle Masses. R. E. Rewell.

Brit. M. J. **484**: (Oct. 16) 1943.

The concentration of potassium is much greater in cells than tissue fluids. Eichelberger (1941) found 25 times as much by weight in dog's muscle (corrected for blood and fat content) as in the serum. This great difference in potassium concentration on the two sides of the cell membrane is maintained only so long as this structure remains undamaged. Prolonged asphyxia is one method of causing such damage. Baetjer (1935) found, by measuring its concentration the perfusate, that loss of potassium from cat's muscle increased as the rate of blood-flow was reduced, but that the actual excitability of the muscle remained unaltered. Fenn et al. (1939) found that there was a limit to the potassium reduction in cat's muscle that could be brought about by perfusion. Later, Fenn, Koenemann, and Sheridan (1940) observed that no increased loss of potassium or production of lactic acid followed periods of asphyxia in perfused frog's legs.

A series of observations is reported in which the serum potassium showed a significant rise after the removal of a tourniquet applied to a limb during the course of orthopaedic operations.

The extra potassium in the circulation might have come from the cells in the ischaemic area or from the liver by the action of hypothetical substance released in the limb.

SCHOOLS APPROVED FOR TRAINING PHYSICAL THERAPY TECHNICIANS

By the Council on Medical Education and Hospitals of the American Medical Association *

Name and Location of School	Medical Director	Emergency Course: Entrance Requirements †	Emergency Course: Length in Months	Emergency Course: Classes Start	Emergency Course: Tuition	Emergency Course: Certificate, Degree, Diploma	Regular Course: Length in Months	Regular Course: Classes Start	Regular Course: Tuition	Regular Course: Certificate, Degree, Diploma
Army and Navy General, Hospital, Hot Springs National Park, Ark.	Capt. Walter J. Lee, M.C.	b	6	Oct	None	Certificate	....	.....	.....	.....
Children's Hospital, Los Angeles[1]	Steele F. Stewart, M.D.	a-b-c	6	FebAug	$200	Certificate	12	FebAug	$200	Diploma
College of Medical Evangelists, Los Angeles[1]	Fred B. Moor, M.D.	a-b-c	....	.....	.....	.....	12	JanJuly	$200	Certificate
University of California Hospital, San Francisco[1]	Frances Baker, M.D.	a-b-c	....	.....	.....	.....	12	Feb	$150	Certificate
Stanford University, Stanford University, Calif.[1]	Wm. H. Northway, M.D.	a-b-d[2]	7	Quart.	$286	Certificate	10	Quart.	$401	Cert. or Degree
Fitzsimons General Hospital, Denver	Maj. O.L. Huddleston, M.C.	b	6	Oct	None	Certificate	....	.....	.....	.....
Walter Reed General Hospital, Washington, D. C.	Capt. D. L. Rose, MC.	b	6	Quart.	None	Certificate	....	.....	.....	.....
Northwestern University Medical School, Chicago	John S. Coulter, M.D.	a-b-d	....	.....	.....	.....	9	JulyOct	$200	Certificate
State University of Iowa Medical School, Iowa City	William D. Paul, M.D.	b-c	6	MarSept	None	Certificate	9	MarSept	None	Certificate
Bouve-Boston School of Physical Education, Boston	Arthur L. Watkins, M.D.	a-b-c[6]	6	June 26	$250	Certificate	3-4 yrs.	Sept	$400 yr.	Dipl. or Degree
Harvard Medical School, Boston	Frank R. Ober, M.D.	a-b-c	6	Mar 15	$250	Certificate	9	March 15	$300	Certificate
Boston University Sargent College of Physical Education, Cambridge, Mass.	Louis Howard, M.D.	a-b-c	....	.....	.....	.....	24	JanOct	$435 yr.	Cert. & Degree
University of Minnesota, Minneapolis[1]	Miland E. Knapp, M.D.	a-b-c[3]	....	.....	.....	.....	12	Mar	$112[4]	Certificate
Mayo Clinic, Rochester, Minn.[1]	Frank H. Krusen, M.D.	a-b-c	6	Jan-July	None	Certificate	9	JanJuly	None	Certificate
Barnes Hospital, St. Louis	Frank H. Ewerhardt, M.D.	a-b-c	....	.....	.....	.....	9	Oct	$200	Certificate
St. Louis University School of Nursing, St. Louis[1]	Alexander J. Kotkis, M.D.	HS	....	.....	.....	.....	4 yrs.	JanSept	$250 yr.	Cert. or Degree
O'Reilly General Hospital, Springfield, Mo.	Maj. John H. Aldes, M.C.	b	6	Oct	None	Certificate	....	.....	.....	.....
Hospital for Special Surgery, New York City[1]	Kristian G. Hansson, M.D.	a-b-c	....	.....	.....	.....	9	Sept	$300	Diploma
New York University, New York City[1]	William Bierman, M.D.	a-b-c	....	.....	.....	.....	9	FebSept	$396	Cert. & Degree
Cleveland Clinic Foundation Hospital, Cleveland	Walter J. Zeiter, M.D.	a-b-c	....	.....	.....	.....	9	Sept	None	Certificate
D. T. Watson School of Physiotherapy, Leetsdale, Pa.[1]	Jessie Wright, M.D.	a-b-c	6	JanJuly	$200	Diploma	12	July	$200	Diploma
Graduate Hosp. of the Univ. of Pa., Philadelphia[1]	Wm. T. Johnson, M.D.	a-b-c	....	.....	.....	.....	12	Sept	$200	Certificate
Brooke General Hospital, San Antonio, Tex.	Maj. Albert Loiselle, M.C.	b	6	Oct	None	Certificate	....	.....	.....	.....
University of Wisconsin Medical School, Madison	F. A. Hellebrandt, M.D.	a-b-c[1]	6	OctApril	$72[4]	Certificate	9	OctApril	$96[4]	Certificate
Richmond Professional Institute, Richmond, Va.	T. W. Wheeldon, M.D.	a-d[5]	....	.....	.....	.....	9-12	Sept	$200-220	Certificate
Duke Hospital, Durham, N. C.	Lenox D. Baker, M.D.	a-b-c[1]	....	.....	.....	.....	9	Sept	Univ. fees	Certificate

† Courses are so arranged that any of the entrance requirements will qualify students for training. a. = Graduation from accredited school of nursing; b = Graduation from accredited school of physical education; c = Two years of college with science courses; d = Three years of college with science courses; HS = High school graduation.

1. Male students are admitted.
2. High school graduates accepted for a four-year course leading to A.B. degree; students admitted quarterly and tuition is $143 per quarter.
3. Medical technology graduates with B.S. degree also admitted.
4. Nonresidents charged additional fee.
5. Those with degree from other colleges also accepted.
6. High school graduates admitted to regular course.

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